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Minnesota Medicine

Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society

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Contents

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WHITE BILE

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"WHITE bile" is a misnomer, but it designates so quickly what otherwise would require a rather cumbersome designation that it appears to stand. The fluid encountered in these cases is at times whitish, but even more often a thin colorless liquid like water; so much so that one writer[3] states that at the first moment on aspirating from the hepatic duct he thought the aspirated liquid was cerebro-spinal fluid. The so-called white bile is, therefore, often not white and it is not bile. However, the fluid is in the place where normally bile is found. Hydrops of the gall bladder is not considered here. It is a relatively trivial condition. We are only discussing the much more serious cases where the bile *ducts* contain the mentioned fluid.

"White bile" is greatly dreaded by the surgeon because it tells us that the liver has stopped its normal excretory action. Bile is partly an excretion and partly a secretion. The liver function is complicated and manifold. Life may go on for a considerable length of time in a case of icterus though the bile channels be tightly blocked, because the production of bile goes on with the bile being discharged into the blood. In such a case it is excreted at least partly with the urine and a little through perspiration. But white bile is also encountered in cases where the liver has ceased functioning or at least a high degree of functional disturbance has been reached. This is particularly seen in some cases of general overwhelming sepsis where white bile is at times encountered without any demonstrable obstruction of the bile channels. The cause of white bile in these cases is a grave toxic damage to the liver parenchyma which has become too disintegrated for bile production. The patient dies of sepsis without or with only moderate icterus, and it is thus merely a finding at the autopsy overshadowed by devastating septic processes in other parts of the body.

LaManna[4] reported in 1936 nine cases of white bile from the pathological institute of the University of Berlin. Of these nine cases four had complete obstruction and severe icterus, while the other five were varied forms of sepsis with no obstacle in the bile channels. It is noteworthy that out of these five septic cases with no obstruction to the flow of bile, four were babies of from two and a half weeks to two years of age. In the case of the newborn this may be explained by the fact that the liver at birth is easily injured when exposed to unduly violent forces acting on this delicately structured and at the same time very bulky organ. Add to this a sepsis usually starting from the navel (umbilical vein), and the occurrence of disintegration of the liver parenchyma in such babies is readily intelligible.

It is important to differentiate between these two separate and distinct types in the white bile cases. The one is due to sepsis which disintegrates the liver parenchyma and causes a true cessation of any bile secretion, a true acholia. These cases are only seen on the autopsy tables, and the icterus is not deep, nor a prominent feature in the picture. The second type is due to obstruction, where the bile producing ability of the liver is only hampered but not destroyed

except in the long end. The bile is discharged partly, and under difficulty, into the blood stream. This form of white bile is therefore not due to a true acholia but to a paracholia.

The white bile with which the surgeon comes in contact is caused by obstruction. The fundamental reason for the occurrence of this quite rare condition is not well understood inasmuch as severe obstructive icterus with dark bile is so often seen. Does white bile occur only after a great length of time during which obstruction is present? This explanation is not satisfactory and experiments on animals contradict it. Furthermore we see quite often cases of long standing with a regular olive colored skin come to operation, but white bile is rarely seen.

Among 1,064 operations on the biliary passages in my private practice only nine cases of white bile were encountered, and one of them does not even strictly belong here as we shall see later.

While earlier authors had seen white bile only in obstruction by tumors this had to be revised later on. Malaguti (cited by LaManna) collected 143 cases of white bile and found stones as cause of the obstruction in 46.8 per cent, while only in 34.5 per cent neoplasms. Some cases had only localized inflammations, stenosing scars or tuberculous portal lymph nodes or even only kinks in the extra-hepatic channels. Many reported surgical cases of white bile had an obstruction more or less complete, and especially for a considerable time. Have we then a gradually developing exhaustion of the liver, leading to a similar end result as in the rapid exhaustion by overpowering sepsis? No. This cannot be at the bottom of the condition if stated in a sweeping way, because in the majority of the observed cases, the liver resumed a quite normal secretory activity within a day or even less after the obstruction was removed. The parenchyma had only been hampered mechanically from expelling its secretion through normal channels. Notwithstanding continuous elimination of bile through the urine the icterus had stayed at its height or appeared to become even more intense before the operation; the liver, therefore, must have continued some of its activity up to the time of operation. Before operation we have no means of finding out if there is white bile, and therefore, most always we do not know how long the contents of the ducts have been "white."

Rous and McMaster, Bernhard and Aronson experimented on animals, where, by ligation of the ducts, white bile was at times observed. The earliest appearance of white bile is found in the reports of Rous and McMaster where this change occurred as early as after eight days while Valdoni (LaManna) and Aronson[1] saw it only after two to four months in dogs. Mostly it took at least three or four weeks as I deduce from the reports. In one of our cases (No. 6) it took less than two weeks after the first signs of icterus. The liver need not be very severely damaged. So one reads in Bernhard's article[2] that dextrose and galactose tolerance tests showed no perceptible damage to this part of liver function. However, we have clinically different outcomes. There are the favorable ones with prompt resumption of apparently all the liver function and full recovery. On the other hand, when bile of normal color does not come forth at the end of thirty-six hours and especially after forty-eight hours, the prognosis becomes very serious. I have seen recovery in a case where proper bile appeared as late as thirty-six hours after draining the duct, but another case of mine died after four days with no colored bile showing up. Judd saw normal bile appearing even after four days. Some earlier authors thought that white bile was due to a reflux of pancreatic secretion. The only authentic case of this kind I happen to know of was in my own practice and will be reported below: but, to say it beforehand, the condition was something entirely different from that under discussion.

Why should the contents of the bile channels become entirely colorless? At the time when obstruction occurs the ducts must have been engorged with normal, probably dark bile. Where does the coloring matter go? Was it absorbed or disintegrated, or both? Bernard accuses the leukocytes for the disintegration, but this is untenable where the contents are perfectly water-colored as in a case reported for instance by Lake of London and in one of ours which will be discussed. Aschoff and LaManna, as pathologists, and many clinicians following Naunyn give infection the first place as the underlying cause for the formation of white bile. In their book on "Diseases of the Gall Bladder and Bile Ducts" (1940) Walters and Snell[9] say on Page 47, "White bile . . . probably represents a true pathologic biliary hypersecretion."

It seemed of interest to give this picture of divergent views as one encounters it in the surgical literature where the numerous and classical experiments of Rous and McMaster are apparently not properly considered. Rous and McMaster[7] have cleared up the problem in a thorough manner and have proven that infection plays no role or only a very secondary one. They produced the picture of white bile in dogs, cats and monkeys under perfectly aseptic conditions where plate cultures remained sterile. They saw all stages of alteration from green to colorless bile. When the gall bladder was left in communication with the tied off main bile duct, absorption of fluid could take place in the gall bladder and the bile became inspissated. A certain amont of bile secretion into the blocked ducts was therefore possible for a longer time than in experiments where the gall bladder was removed or excluded otherwise. When the resorptive action of the gall bladder persisted the bilirubin changed into the much darker biliverdin by oxidation which (at least partly) simulated a concentration. The question naturally comes to you: Why is white bile observed so rarely in our surgical work while severe obstructive icterus of long standing is seen so frequently? Perhaps general constitutional differences as well as local factors come into play. The answer would therefore be purely speculative at the present time. But as to the origin of colorless bile in obstruction of the ducts McMaster and his co-workers have definitely settled the question. They ligated ducts above and below; the isolated segment became distended with a thin colorless fluid, watery or thinly mucinous. Cultures were sterile. The segment became greatly distended. This secretion, therefore, was going on notwithstanding considerable intra-ductal pressure. A strip of duct 2 cm. long between the two ligations yielded 8.5 c.c. of fluid in six days, surely a surprising amount. In eleven dogs with total obstruction lasting eight to twenty-seven days the contents of the main channels were entirely colorless. The same was observed on cats after eleven to fourteen days. Cholesterol was practically absent. This disappearance of the originally present bile cannot have been caused in such short a time by disintegration or by leukocyte activity as some authors contend. On the other hand an absorption of bile salts, pigment or cholesterol does not occur even in the gall bladder. Where a complete blocking is caused by some absolute obstacle there must be a place of escape for the originally present real bile. It must be forced out somewhere (and at the same time gradually diluted) by the colorless thin secretion of the bile channels. It is of interest that already long ago (in 1868) Heidenhain made physiological studies on absorption from the bile channels and the escape of bile in obstruction (Rous and McMaster, also Herrman, "Physiologie"). He located the site of escape of entrapped bile at the junction of the intralobular bile capillaries with the collecting ductlets in Glisson's septa. Buerker finds it practically at the same place, i.e., in the peripheral area of the liver lobules. It stands to reason that when the biliary secreting pressure is exceeded by that of the ducts, the very delicate bile capillaries must give way.

I should consider the happenings to be about as follows: The obstruction in a case of carcinoma develops gradually. The pressure in the ducts increases and, as Rous and McMaster have shown, the liver secretion becomes poorer in the essential ingredients. As the biliary secretory pressure is quite low (24 mm. H_2O) the flow of bile ceases at a moment when the pressure in the ducts exceeds this point. The ducts with their higher secretory force still go on pouring out their colorless fluid. With this increased pressure the contents of the bile ducts can still be forced through the obstacle and thus the contents of the channels are gradually thinned out and become paler and paler until complete stoppage takes place. Where sudden complete obstruction occurs as in the case of calculi, absorption at the periphery of the liver lobules will have the same effect of a gradual dilution and finally replacement of bile by the colorless secretion from the ducts. The two modes of exit may of course be combined in some cases.

My experience with white bile as mentioned before is limited to nine cases. Four of them offer nothing of further value than that in two of them normal bile appeared on the evening of the day of operation, in another after twenty-four hours, and in the fourth one after thirty-six hours. They recovered fully. A fifth one died of cholemia after barely four days without return of normal bile, not even the slightest coloring.

Case 6.—Mr. J. B. H., a printer, sixty-five years old, gave in short the following history: He had suffered from no serious illness prior to the present one which

had started in the later part of June, 1939, with epigastric pain, vomiting and diarrhea. His surgeon sent him to the hospital on August 9 for closer study. The leukocytes were 11,350 on August 10 and six days later came down to 8,000. Throat, chest, abdomen and urine gave no further clue. An x-ray study of the gall bladder revealed a very large gall bladder with poor concentration of the dye. There was no jaundice.

He was operated upon August 19. A large, very tense congested gall bladder was found which had to be aspirated before it could be gotten hold of. One hundred c.c. of tarry bile were evacuated in this manner. Stomach and duodenum were normal. The gall bladder was removed. No stones were present. The cystic duct was small. Palpation of the deeper ducts revealed nothing further. The thickened appendix was then taken away and the abdomen closed. The pathologist's report reads (in part): Gall-bladder wall considerably thickened. A few lymphocytes under the epithelium and in muscularis: chronic cholecystitis. The operative course at first was very smooth. The temperature after a slight rise on the day following the operation never reached higher than 98.6 degrees, and on August 28 the patient was allowed to leave the hospital. At home he did not do well. The old epigastric distress persisted and gradually increased. When a slight jaundice became evident his surgeon had him readmitted to the hospital on September 11. The leukocytes were 7,750 and the hemoglobin 81 per cent. The roentgenologist was unable to demonstrate any abnormality in the region of the papilla of Vater. I was then asked to see the patient in consultation on September 16, 1939. He was cachectic looking and emaciated. The feces were clay-colored, and the dark urine contained much bile. The bleeding time was 3.5 minutes and coagulation time 6.5 minutes. As the icterus was increasing and the sensorium had become somewhat blurred, operation without much delay was necessary. On September 18, thirty days after the first operation, we reopened the abdomen under spinal anesthesia. After dividing the adhesions we noticed in the region of the hepatic duct a bulging which fluctuated and was of the size of a distended jejunum. Through a fine needle 85 c.c. of a very thin water-colored fluid were withdrawn. Having only a 20 c.c. syringe at hand the needle punctured the wall four times which was undoubtedly the reason we later found a few blood cells in the centrifuged liquid. An incision was made and more fluid removed. The inside of the duct was smooth and entirely white, in striking contrast with all the bile-stained surrounding tissues. A curved uterine sound was worked around some folds upward and finally entered the right and then the left hepatic duct. We could now feel certain that we had to deal with an enormously dilated hepatic and common duct. But we were not able at first to enter far downward on account of a number of folds which had formed after the evacuation. With blunt artery clamps these folds were then taken hold of as they came into view. Crawling thus downward from fold to fold on the inside of the duct the lower portions of it became stretched and finally the probe under some force entered the duodenum. Though suspecting it, we could not make out a definite sign of neoplasm. Immediately upon withdrawal of the probe a small caliber catheter was introduced and pushed some inches through the papilla. In addition a medium sized catheter was laid into the duct which was then closed around the two tubes with fine catgut. A collapsible rubber drain was placed in the neighborhood of the opeing in the duct, and the abdomen closed.

The postoperative course was afebrile. The pain was temporarily relieved, and with the rapid decrease of the jaundice, the sensorium became more free. Six days after the operation the catheter which led through the ampulla was removed and on the eleventh day the tube leading into the dilated duct was withdrawn. Bile then escaped profusely for a few days, but soon ceased. The patient died, however, after about five weeks and the autopsy revealed a carcinoma of the head of the pancreas.

Case 7.—This was similar to the foregoing case. However, the great enlargement of the duct had existed already at the time of the first operation and as the gall bladder containing no stones did not appear changed apart from being large, it had not been removed. The duct was the size of a man's wrist according to the surgeon's report. The operation had taken place on August 14, 1939. On incision "a very large amount of white bile gushed out. What came first was clear-like water but was followed by more whitish fluid. Similar fluid came forth when the gall bladder was compressed." Under considerable difficulty a probe could be inserted through the ampulla, which the surgeon then dilated. No pathology could be made out and the pancreas gave no evidence of a tumor. The duct was drained. Before the abdomen was closed, colored bile appeared. The patient was discharged on September 11. She was readmitted on October 6 as she suffered from a great deal of pain and was steadily losing ground. When I first saw her on October 30, she had vomited practically everything for many days and had been kept alive only by repeated blood transfusions and glucose intravenously. X-ray examination demonstrated a complete duodenal obstruction low on the descending portion. The only chance was a gastro-enterostomy which was made, and at that time a large mass in the pancreas was felt. But the patient succumbed a few days later from exhaustion. The diagnosis of carcinoma of the pancreas was confirmed at autopsy.

In this case there had been a free communication between the gall bladder and the common duct, which Rous and McMaster found to delay the formation of white bile because the gall bladder has marked absorbing qualities and thus takes care of part of the over-pressure. This could delay the formation of white bile, but it did not prevent it.

Rous and McMaster's experiments showed that

if the common duct was obstructed for twelve to fourteen days with a normal gall bladder present, green fluid was found in the ducts. If the gall bladder was functionless or had been removed, they found white bile to have formed after the same length of time, i.e., twelve to fourteen days. Where white bile was encountered in the presence of a gall bladder, these authors consider the gall bladder to be functionless like a simple diverticulum of the ducts.

Case 8.—In this case I only had the opportunity to see the autopsy. The gall bladder had been removed nineteen and one-half years previously. Death had followed one month after an operation of draining the common duct in severe icterus. White or rather absolutely colorless bile had been aspirated from the duct which was of 1-inch diameter. The autopsy revealed a constricting carcinoma of the pancreas. There were no signs of peritonitis. The interesting feature was that though normally colored bile appeared thirty-six hours after the operation, and the bile fistula was kept open, the severe icterus did not recede. There was a reasonable output of bile, up to 550 c.c. in twenty-four hours, a rather small quantity for a muscular man who took nourishment. The amount of bile gradually decreased later on. The liver was not cirrhotic, it was only dark as one would expect. The biliary fistula was wide open and the ducts were freely patent when followed into the liver. But the icterus had remained very deep. We had here, therefore, a degree of damage to the liver through the pre-operatively existing biliary obstruction which almost—but only almost—permitted recovery of the liver function. The condition of the liver, one might say, was on the fence, and gradually began to lean the wrong way. And this notwithstanding reappearance of bile thirty-six hours after relieving the intraductal overpressure.

The next was a very exceptional case, as we were confronted with a spurious form of "white bile." The story is the following:

Case 9.—Mr. J. B. S., forty-one years old, had a posterior gastro-enterostomy for supposed duodenal ulcer in May, 1930. The findings were not entirely definite according to the suregon's statement, and the patient was no better after that. Nine months later, in February, 1931, he came to us with symptoms of great distress which pointed strongly to the gall bladder. An x-ray examination, made elsewhere, found the gall bladder to be "abnormal." On February 10, 1931, the gall bladder was removed. We encountered an undue amount of adhesions. The gastro-enterostomy, we knew from our x-ray study, functioned and showed no flecks. The excised gall bladder on examination disclosed microscopically a round cell infiltration in the muscular coat, quite marked in some areas. Bacteriologically microörganisms of the colon group were cultured from the wall of the gall bladder. Recovery was prompt, but six months after the operation an attack of severe pain came on. It blew over. There was no jaundice, and the patient was then completely well for two years. He was again admitted to the hospital on October 16, 1933. Since August, 1933, repeated attacks (about five in all) of severe pain in the region of the gall bladder had occurred with clay-colored stools, dark urine, moderate but definite jaundice and pruritus. The condition looked like an intermittent obstruction of the common duct due to stone. The note was made that the present attack had started four days before admission with an exacerbation two days later after the noon meal consisting of soup and a sandwich. Since October 5, when he had an attack previous to this last one, jaundice persisted.

On October 18, 1933, we re-opened the abdomen. The temperature on the two preceding days had ranged between 99.5 and 100.5 degrees. Diffuse and widespread adhesions were again encountered. The pancreas was so hard and bulky that it was decided to snip a small piece away for microscopic examination, which revealed, however, a simple inflammatory condition. The common duct was hard and strictured at the place of junction with the cystic duct. It was slit beyond the thickened area at both ends. It was probed into the duodenum, but no stone was found. A simple rubber catheter with an additional hole was inserted into the duct downward toward the duodenum. There was no suturing of the duct. Collapsible rubber drains were placed near the duct area and the abdomen closed. The patient was turned flat on his stomach for several days. The temperature which rose on the next day to 101.4 degrees came gradually down to normal in the course of a week.

Practically all bile came through the catheter. The itching had disappeared after forty-eight hours, and five days after the operation the jaundice had decidedly receded. On the morning of the sixth postoperative day, October 24, the discharge through the catheter had changed suddenly. The receiving bottle contained a water-colored, transparent but quite viscid secretion apparently containing mucin. When we first saw it, about four hours after the bottle had been emptied of normal-looking bile, 120 c.c. of this colorless fluid had gathered. This sight was, of course, for a moment perplexing as white bile would mean a serious damage to the liver. The dressings had just been changed by the nurse. We nevertheless wanted to inspect the wound, and to our great relief found the inner dressings soaked with golden yellow bile which escaped from the wound along side the catheter. There could be only one explanation. The clear and colorless fluid from the catheter was immediately tested for amylase, lipase and protease. Within perhaps ten minutes the starch was converted into sugar. In twenty-four hours the butter was decomposed into crystals of fatty acids with yellow oily globules floating on top, and hashed beef meat was dissolved. Thus we had a complete separation of pancreatic secretion from the bile. By evening of the same day 360 c.c. of the same limpid fluid without even a trace of bile color were discharged into the bottle in

about twelve hours while normal bile drained profusely into the dressings. The patient had eaten a very moderate amount of the general hospital diet for lunch, but had had a good breakfast. The catheter was then removed. Ten days after, there was some bile found in the stool. On November 2, fifteen days after the operation, the patient left the hospital in very good spirits with the fistula closed. He has felt well and works every day ever since according to his very recent report.

The observation that 360 c.c. of pure pancreatic secretion were collected in twelve hours, from 6 A.M. to 6 P.M. from a medium sized rather strong man, is of physiologic interest. Such opportunities for measuring are rare.* The obstruction to the flow of bile along the catheter was found to be due to a very marked swelling of the segment of the catheter which had been in the duct. On withdrawal of the tube, this portion was black and formed a club-shaped thickening, due perhaps to poor rubber (?).

The spurious "white bile" which we encountered in this patient was water-colored and could be called practically limpid. It was rather thickish on shaking, which is not ordinarily seen in white bile. It was quite different from the findings in the other cases.

Examination of the "White Bile"—Case 6

After preservation on ice for a number of days the fluid became a trifle opalescent and syrupy but at the time of operation it had been colorless and thin and was readily aspirated through a very fine needle.

Specific gravity—1007
Reaction—Faintly alkaline
Centrifuged sediment—About 10 red blood cells per field and an occasional white cell.
Albumen—Trace
Globulin—Trace
Urea nitrogen—11.76 mgms. per 100 c.c. of fluid
Urea—25.16 mgms. per 100 c.c. of fluid
Non-protein nitrogen—25.86 mgms. per 100 c.c. of fluid
Mucin—Trace
Bile acids—None
Bile pigment (Bilirubin)—None
Carbohydrates—Trace
Calcium—6.0 mgms. per 100 c.c. of fluid
Chlorides—0.813% (as sodium chloride)
Digestive ferments (diastase, lipase, protease)—None.

Albumen and globulin traces can be explained by the admixture of a very small amount of blood from the puncture wounds.

N. C. Lake gives an analysis of the colorless fluid of his case as follows: Protein (albumen and globulin)—none; mucin—none; bile pigments—none; bile salts—none; cholesterol—none; chlorides (reckoned as NaCl) 0.862 per cent. Lake's chemical analysis of the "white" bile in his case is the only one I could find though others may have made similar studies. Reports of chemical analysis of simple hydrops of the gall bladder do not belong to our subject.

The picture of a colorless fluid in the bile channels with a completely white mucosa while all the surrounding tissues are deeply bile stained is really a perlexing sight. But we may well remember that saliva, tears, gastric and intestinal secretions, cerebro-spinal fluid are not stained either in icterus. Catarrhal sputum in a case of icterus is not bile stained while pneumonic sputum is. The first is a secretion while the last is an exudate with much blood and serum in it.

The difference between the septic "white bile" cases without obstruction and little or no icterus on the one hand and on the other the surgical cases with obstruction and deep jaundice is—be it repeated—a fundamental one. But a mechanical obstruction as mentioned before, may not always be very readily demonstrated. As a side remark let me mention that a simple spasm in the ductal system may lead to an icterus. This is demonstrated by what is called "icterus ex emotione." A sudden terrible fear may cause the appearance of jaundice within a few minutes, as for instance in a case of Umber's.[8] It can only be explained that the psychic shock brought on a severe spasm, probably not only in the larger but even in the small bile channels, with a sudden traumatizing blocking and evacuation of the already formed bile into the blood circulation. However, such a condition is too transitory to become a source of white bile.

Historically it is of interest that an excellent description and surgical discussion of our subject came already in 1912, from Lenormant under the title "L'hydropisie de la voie biliaire principale."[5] He reports a case of his own; the patient died of anuria after cholecystectomy and drainage of the hepatic duct. Carcinoma of the ampulla of Vater was the underlying condition. Lenormant

Archives of Internal Medicine, Vol. 61, p. 739, May, 1938. Report of case of complete pancreatic fistula in which the total external secretion amounted to 600 c.c. per day average. In the discussion Dr. W. T. Coughlin gives 400 c.c. per day as the average human secretion. In the same discussion Dr. Rolf Ium reports one patient with 200 c.c. per day, another with 500 c.c. per day, which he considers average secretion, and one patient with 1,400 c.c. per day, the largest amount of secretion yet recorded for any individual. All observers concur that the ingestion of food and liquid and the intramuscular or intravenous injection of secretin increase the rate of pancreatic external secretion.

gives the literature up to that time with a careful analysis of the cases. He found thirty instances including his own. The article closes with the advice not to excise the gall badder in these cases so as to be able to use it for an anastomosis with the intestinal tract in case this should become necessary. Inasmuch as in those days white bile had been seen only in neoplastic obstruction, the suggestion was very apropos.

Summary

1. The white bile as seen by the pathologist in overwhelming sepsis rests on a fundamentally different cause from that seen by the surgeon in cases of obstruction of the bile ducts. The difference is that of acholia versus paracholia. These two conditions have not been kept sufficiently separate in the literature.

2. Infection is not the basic cause in the surgical white bile. The underlying condition is obstruction to the bile flow, though frequently infection is also present.

3. White or rather colorless bile is a secretion of the ducts with their glands. A water-colored secretion from bile-choked liver acini is not really thinkable. On the other hand a poorly-colored or even uncolored bile in overwhelming sepsis without noticeable obstruction is the result of a greatly disintegrated liver though even here some obstructive swelling in the smallest ducts is conceivable as coöperating.

4. The dextrose-galactose tests indicate that in cases of even severe icterus, experimentally provoked by ligation of the common duct, the liver function is not (at least for a long time) destroyed in this respect.

5. Normally colored bile may start flowing immediately after decompression followed by recovery—or may not appear until death.

6. If normal bile does not appear thirty-six or forty-eight hours after releasing of the obstruction, the prognosis becomes very grave though even after four days' delay recovery was observed.

7. One case is reported with chemical analysis of the fluid. In another case of (what looked like) white bile the condition was due to reflux of pancreatic secretion through a catheter in the common duct.

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ABNORMAL UTERINE BLEEDING IN ADOLESCENCE

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DORLAND'S medical dictionary defines adolescence as the period between puberty and maturity. We may include the ages from ten to twenty years in this developmental period. A careful study of the normal and abnormal uterine bleeding in these young women may aid materially in the better understanding of menstruation and its disorders.

The onset of catamenia in this country is expected between the ages of twelve and fourteen. However, the menarche starts between eleven and sixteen years in many normal girls. Race climate, social station and general health influence the menarche, with familial and individual variations. We might speak of premature menarche before the ninth year, delayed menarche after the eighteenth year. A year or more to establish a regular cycle is expected. A regular rhythm of menstruation from twenty-eight to thirty-day cycle with a flow lasting from four to five days is considered ideal. Obviously, such regularity is seldom encountered. A range of from twenty-one to thirty-five-day intervals with

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a flow varying from two to six days are frequent in apparently normal menstruation.

Incidence of Abnormal Adolescent Bleeding

Keene and Payne[13] found that of 500 patients admitted to the hospital for abnormal uterine bleeding only 4 per cent were under twenty years of age. Obviously, the fact that the majority of the cases of abnormal adolescent bleeding do not need hospitalization accounts for this low incidence. Of 7,634 women entering the University of Minnesota from 1938 to 1942, 22 per cent or 1,710 stated that their menstrual cycles were irregular. Of these 731 had had medical care during the high school period, and 225 complained of profuse bleeding. During these four years 2,302 students have consulted the gynecological department of the Student Health Service. It was found that very few women menstruate every twenty-eight to thirty days, most of the cycles occurring from three to five weeks. Vaginal discharge, dysmenorrhea, and irregular bleeding are the common complaints which bring the students in for a gynecological examination. There were 630 women (27 per cent) whose primary complaint was irregular bleeding.

Etiology

Before we consider the causes of abnormal uterine bleeding of the adolescent, we should consider the function of normal uterine bleeding, menstruation. The broad definition of menstruation advocated by Corner[4] and Hartman[12] include all of the factors associated with cyclic bleeding from the uterus. For clinical purposes, however, it is more satisfactory to limit the term menstruation, to "bleeding which occurs from an endometrium which has undergone the cycle of changes necessary for nidation," in accordance with the work of Schroeder[20] and Meyer.[16] Only such an endometrium can be considered normal, since the physiological purpose of the uterus is to prepare an endometrium suitable for the implantation and nourishment of the fertilized ovum. If menstruation is to be considered a normal phenomenon, then it must consist of changes in a normal structure.

The changes of the endometrium are controlled by a complex endocrine mechanism. Bleed-

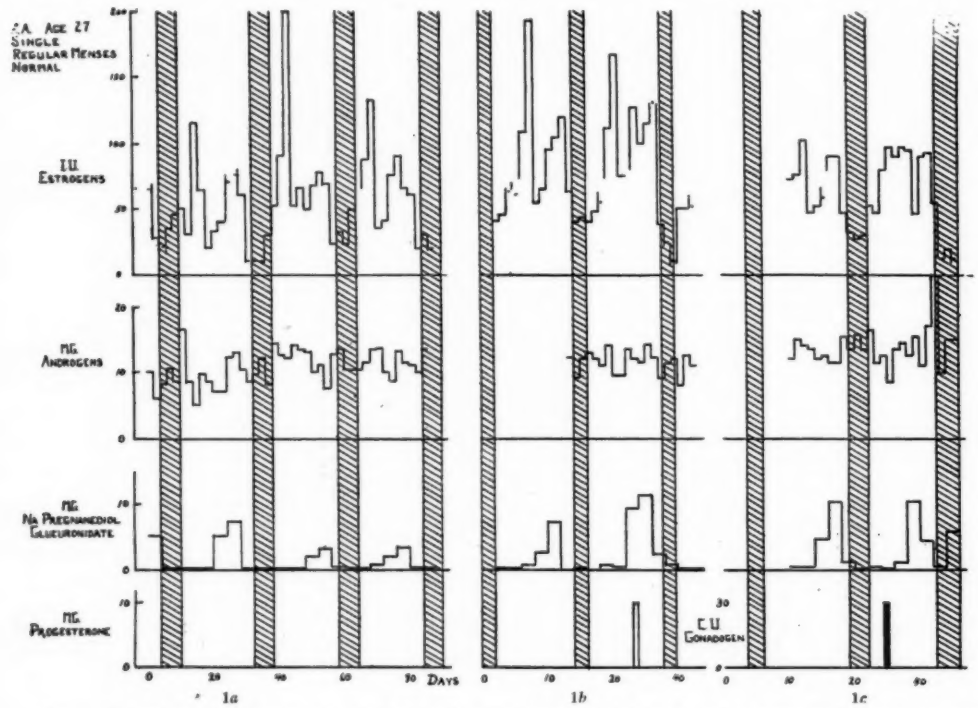

Fig. 1. Normal menstrual cycle. (*a*) Untreated; (*b*) progesterone injections; (*c*) mare's serum injections. Shaded areas indicate menstruation.

ing from an endometrium similar to that seen in normal menstruation can be produced in castrate monkeys and ovariectomized women if the subject is treated with estrogens, followed by progesterone. Bleeding follows in twenty-four to seventy-two hours after the progesterone is discontinued, which is the interval observed in normal menstrual cycles after the disappearance of pregnanediol in the urine (Fig. 1). Gillman[9] has shown that there is an optimal estrogen-progesterone ratio for the normal cycle in women. An ovarian deficiency is the probable cause of abnormal bleeding in the adolescent. This may be primary, or secondary to another endocrine gland, probably the pituitary or thyroid, or to some constitutional disorder. In the primary type, the immature ovary fails to respond to the pituitary stimulation, consequently rupture of the follicle and luteinization may not occur. Three important factors in uterine bleeding are the hormonal effect of the pituitary gland, the response of the ovary, and the response of the uterus to both the pituitary and ovarian stimulation.

Classification of Uterine Bleeding

The literature on abnormal bleeding is confusing because of the discrepancy of terms used. Uterine bleeding may be divided into two main categories: disturbances of menstruation and nonmenstrual bleeding. Uterine bleeding will persist until healing by epithelization occurs. The amount of bleeding depends upon the size of the vessels involved and the resistance to closure by natural or mechanical means.

The Schroeder classification has been used as a basis to sort out the various types of bleeding. As more information is available from hormone studies and endometrial biopsies, the subdivisions of the following classification become apparent:

A. Disturbances of Menstruation.

I. *Disturbances in the regularity of the cycle.*

a. Shortened cycle (polymenorrhea).
   1. Premature interruption of the cycle.

b. Lengthened cycle (oligomenorrhea).
   1. Persistent corpus luteum.
   2. Hyperplasia of the endometrium.
   3. Inhibited cycle followed by a normal cycle.

c. Amenorrhea.
   1. Physiological.
   2. Primary amenorrhea.
   3. Secondary amenorrhea.

d. Acyclic bleeding (metrorrhagia).

II. *Disturbances in the amount of menstrual flow.*

a. Profuse flow (hypermenorrhea).
   1. Hypoplastic uterus.
   2. Hypothyroidism.
   3. Tumors (polyps, submucous myomata).
   4. Pelvic congestion.
   5. Endometrial hyperplasia.

b. Scanty flow (hypomenorrhea).
   1. Hypogonadism.
   2. Hyperthyroidism.

III. *Disturbances in the duration of menstrual flow.*

a. Lengthened bleeding period (menorrhagia).
   1. Irregular shedding and irregular ripening.
   2. Endometrial hyperplasia.
   3. Inflammatory.
   4. Tumors (benign or malignant).

b. Shortened bleeding period.
   1. Hypogonadism.

B. Nonmenstrual Bleeding.

I. *Disturbances under ovarian control.*

a Cystic glandular hyperplasia.
   1. Granulosa cell tumors.

b. Ovulation bleeding.

c. Anovulatory bleeding.

d. Following hormone treatment.

II. *Disturbances not under ovarian control.*

1. Uterine, acyclic bleeding (metrorrhagia).
   a. Tumors (polyps, myomata).
   b. Incomplete abortion.
   c. Infection.
   d. Malignancy.

2. Adnexal.
   a. Tumors.
   b. Pelvic inflammatory disease.

3. Blood dyscrasias.
   a. Purpura hemorrhagia.
   b. Leukemia.
   c. Aplastic anemia.

4. Constitutional diseases.
   a. Congenital lues.
   b. Tuberculosis.
   c. Cardiac decompensation.
   d. Grave's disease.
   e. Nervous disorders (shocks, fright).

Diagnosis and Pathology

A careful physical examination should be done including a rectal examination, blood studies (including bleeding time and platelet count and Wassermann test) and basal metabolism rate and at times an endometrial biopsy. We may divide the abnormal bleeding into two groups: (1) disturbances of the endocrine glands and (2) bleeding associated with constitutional diseases.

Disturbances of the Endocrine Glands

(a) *Pituitary Dysfunction.*—Many believe that hyperfunction of the pituitary is largely responsible for the menstrual irregularity. Fluhmann[8] described "Pubertas præcox," uterine bleeding before the age of nine, and assumed it to be due

to a hyperfunction of the anterior pituitary where no other pathology could be found. Pituitary dysfunction is also suspected in the obese girl with a normal basal metabolism. The pituitary tion of the pituitary with failure of progestin formation, and resulting in a hyperplasia of the endometrium. In hormone studies the estrin output is maintained at a high level (Fig. 2*a*).

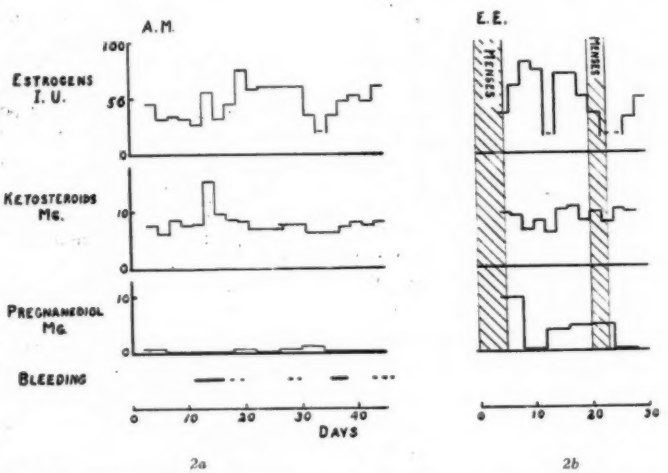

Fig. 2. (*a*) Hormone determinations in the urine in a case of hyperplastic endometrium with severe bleeding. Note the maintenance of a high estrin level. (*b*) Hormone determinations in the urine in a case of irregular shedding of the endometrium. Notice the continuance of pregnanediol secretion throughout the menses.

type is more difficult to recognize in the adolescent than in the adult where all the characteristics of dyspituitarism are evident.

(b) *Hypogonadism.* — The underdeveloped ovary fails to respond to pituitary stimulation. Physical examination reveals an undernourished, tall, hyposthenic girl with underdeveloped secondary sex characters, such as scanty pubic hair, hypoplastic genital organs and underdeveloped breasts.

(c) *Hypothyroidism.*—The characteristics of hypothyroidism may be present, such as obesity, dry skin, fatigue, sometimes mental dullness, and an increased serum cholesterol concentration. However, a normal-appearing girl with a basal metabolism rate slightly below normal is frequently encountered with profuse uterine bleeding.

Pathology of the Endocrine Group.—Cystic glandular hyperplasia is found in adolescence as well as in the menopause. Perhaps for sentimental fear of rupturing the hymen, a diagnostic curettage is seldom done in puberty; however, a severe hemorrhage or prolonged flow during the adolescent period may necessitate it. Cystic glandular hyperplasia is probably due to a dysfunc-

Frequently, the amount of bleeding is not in proportion to the degree of hyperplasia of the endometrium. Gillman has shown the existence of estrogen-sensitive and estrogen-resistant women. The pathology is the same as that found in the adult. The ovaries show many follicles in various stages of growth and atresia with no evidence of recent ovulation or corpus luteum formation.

Irregular Shedding and Irregular Ripening.—Traut and Kuder[22] have shown that whereas hyperplasia represents the Graafian follicle phase, irregular shedding represents the corpus luteum phase. They also describe "irregular ripening," in which the endometrium shows both proliferative and secretory phases. The menstrual flow may persist for eight to twenty-one days. The normal tempo of the onset of the flow is maintained but regeneration of the endometrium is prolonged. To be sure of a diagnosis, a curettage should be done between the eighth and the tenth days of the bleeding. The transition between the functioning stage, proliferation and regeneration will be found in the endometrium indicating a delay in healing. However, in the menarche, the condition is frequently self-limited and curettage

is not necessary. Hormone assays in irregular shedding indicates the excretion of progesterone continuing during the bleeding phase (Fig. 2*b*).

Anovulatory Bleeding.—The discovery of anovulatory bleeding in monkeys indicates that estrin alone might account for menstruation. Markee[14] transplanted small pieces of endometrium in the anterior chamber of the eye in monkeys. He found no difference in the process of menstruation whether ovulation took place or not. He concluded that the actual cause of the uterine bleeding was due to the regression of the endometrium. An increased fragility of the capillaries followed the marked coiling of the spiral vessels. A disproportion between the length of these vessels and the thickness of the endometrium indicated that menstrual flow occurred whenever a regression of the uterine mucosa took place. According to Markee, progestational stimulation of the endometrium was not necessary for menstruation.

On the other hand, Daron[5] found the endometrium to differ in anovulatory bleeding from that of normal menstruation. In the former, the coiled arteries were not at the surface and the terminal arterioles were intact, whereas, in the ovulatory menstruation the surface arteries were completely occluded. Robert Meyer[17] has shown that anovulatory bleeding differs in essential details from menstruation, and indicates an interruption of the menstrual cycle. The regression of the follicle while the endometrium is proliferating is the essential feature of anovulatory bleeding according to Meyer. That anovulatory bleeding is not the general rule in the menarche has been shown by the number of pregnancies reported in countries where child marriages are common. In any case, premature interruption of the cycle is frequent in the adolescent period.

Endometrial biopsies may aid in the diagnosis of abnormal bleeding if it is possible to interpret the endometrial changes in terms of endocrine function. Vaginal smears are simple office procedure but again the interpretation is difficult. In recent years, hormone assays of the blood[7] and urine[24] have also thrown light on the endocrine background in menstrual disorders. Unfortunately, hormone assays are not available to the majority of clinicians as yet, because of the expense and time consumed in running the assays. A single assay is of very little value.

BLEEDING ASSOCIATED WITH CONSTITUTIONAL DISORDERS

(a) Granulosa cell tumor of the ovary should be ruled out by vaginal or rectal examination. Early development of secondary sex characters and menstruation may be due to an excess of estrogen from the ovarian tumors.

(b) In hydrocephalus, tumors of the hypothalamus, and in inflammatory conditions such as encephalitis and meningitis prepubertal bleeding has been reported.[8]

(c) Blood dyscrasias.—Buxton[2] reported seven cases out of 108 of Purpura hemorrhagica where the chief complaint was profuse uterine bleeding, two cases with aplastic anemia and one of acute lymphatic leukemia.

(d) Congenital lues.
(e) Tuberculosis.
(f) Grave's disease.
(g) Cardiac decompensation.
(h) Pelvic inflammatory disease.
(i) Malposition of the uterus.
(j) Teratoma and sarcoma.
(k) Polyps.
(l) Nervous disorders, shocks and fright.

Treatment

A careful examination to determine the cause of the abnormal bleeding should be the first step in the treatment. Until we better understand the endocrine function in respect to the pituitary stimulation and the response of the ovary and uterus, we will continue to treat abnormal bleeding empirically. In the adolescent, particularly, abnormal bleeding is frequently self-limited and is best left untreated. The hormones act as regulators or intermediates in the complicated chemistry of the body. Hormone therapy should be used cautiously unless adequate control studies are made.

Dessicated thyroid extract is indicated when the serum cholesterol concentration is increased or the basal metabolic rate is decreased. Clinically, excellent results have been obtained in profuse menstrual bleeding with small doses of thyroid. Cooke[3] advises thyroid for a period of six months. Plass[18] has also called attention to the use of thyroid when the basal metabolism rate is between zero and minus 10 per cent. Careful observation is necessary because of the variability in the tolerance of the drug. The appearance of palpitation, headache and nervousness in-

dicate an overdose. Small amounts of Lugol's solution occasionally regulates prolonged menstrual bleeding.

The use of estrogens to stop excessive uterine bleeding has been advocated by Hamblen.[11] The ability of estrogens to tone up the uterine musculature and the blood vessels may account for the good results obtained. An endometrial biopsy or hormone assays should precede its use. It is indicated in bleeding from an estrogenic endometrium, atrophic endometrium with a hypoplastic uterus, or when excessive progestational reaction exists. Estrogens should not be given in large doses over a long period of time in the adolescent because they do not stimulate the growth of the ovaries but actually inhibit the pituitary and thus reduce the ovarian function as was brought out by Sevringhaus.[21]

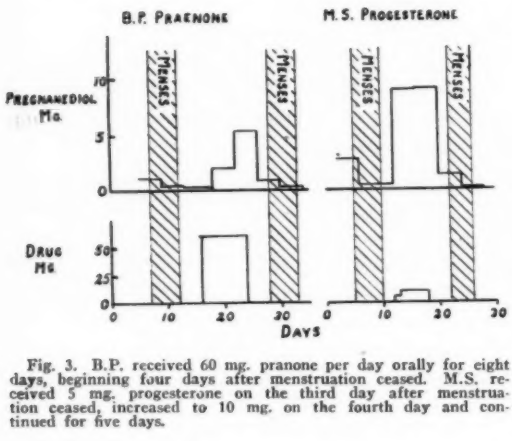

Fig. 3. B.P. received 60 mg. pranone per day orally for eight days, beginning four days after menstruation ceased. M.S. received 5 mg. progesterone on the third day after menstruation ceased, increased to 10 mg. on the fourth day and continued for five days.

Progesterone also has been used in the treatment of abnormal uterine bleeding. Weisbader[23] reported twenty cases of juvenile uterine bleeding treated with oral pregneninolone, the effective dose ranging between 280 and 350 mg. given in divided doses of 50 mg. daily. Three patients who did not respond to the oral therapy were given 25 to 50 I. U. of corpus luteum hormone by injection with good results. Paradoxically, the administration of progesterone is followed by bleeding in the normal subject. If progesterone is administered to a normal woman during the first half of her menstrual cycle, bleeding will occur from a secretory endometrium within the usual interval after the hormone treatment is stopped, according to Gillman and others. Two cases in our hormone study were given progesterone in the first half of the cycle, one receiving 10 milligrams daily by injection and the other was given a daily dose of 60 milligrams of pregneninolone (pranone). Both of these started the uterine bleeding from forty-eight to seventy-two hours after the hormone was discontinued (Fig. 3).

Chorionic gonadotropin has given good results in a small series of cases of profuse uterine bleeding in the adolescent. The dosage is difficult to calculate since we cannot measure the normal output nor the deficiencies present. Gonadotropins should not be given over long periods of time because of the possibility of producing refractive or polycystic ovaries. However, with signs of hypogonadism, a therapeutic test of gonadotropins is advisable. Mazer and Israel[15] gave ten consecutive injections of 30 Cartland-Nelson units on alternate days with good results in his four cases of juvenile bleeding.

Sixty-four cases of profuse menstrual bleeding were treated at the University Health Service from 1938 to 1942. Eleven of these were curetted and two were given blood transfusions because of continued profuse bleeding. Forty-six of these were given chorionic gonadotropin (Antuitrin S or A.P.L.) the usual dose being 500 I.U. daily for four doses starting the fifth day of the flow when possible or directly following curettage. Thirty-eight responded favorably, most of them receiving 200 I.U. doses after subsequent menstrual periods. Before 1939, most of the women with profuse or prolonged flow were given Lipo soluble ovarian substance (Sistomensin) during the first three days of the bleeding cycle, with about fifty per cent being benefited.

Equine gonadotropin (gonadogen) was given to seventeen of the college women with improvement in 40 per cent of the cases of prolonged or profuse menstruation. Gonadotropic hormone from pregnant mare's serum given to normal women in the first part of the menstrual cycle increased the output of estrogens in the urine, but did not affect the pregnanediol levels in our series. In one case in which a single peak of estrogen had been present, 30 Cartland-Nelson units of gonadogen were given intramuscularly on four successive days and intravenously on the fifth day. Urine assays revealed a double

estrogen peak after the administration of this hormone.

Rydberg and Pedersen-Bjergaard[19] treated secondary amenorrhea with 3,000 I.U. of equine gonadotropin daily for five consecutive days followed by 1500 I.U. of chorionic gonadotropin on alternate days for three doses. Menstruation occurred about ten days after the last injection. About one-half of the patients treated continued to menstruate regularly after the therapy was discontinued. Many of those treated were confined to bed for a few days because of pain. Enlargement of one or both ovaries was noted in these cases. A laparotomy was performed on one of these women and lutein tumors were found.

Testosterone propionate has been given for profuse bleeding with good reports. One should be especially cautious in its use in women under twenty years of age. It is better not to give testosterone unless one understands its limitations and dangers.

Combined treatment with estrogens and progesterone have been used successfully in amenorrhea and profuse bleeding. In the latter, control of the bleeding must be accomplished first. Injections of 10,000 I.U. of estrogen is given for fourteen consecutive days followed by 5,000 I.U. of estrogen given concurrently with 5 mg. progesterone for the next seven days. No injections are given for seven days after the onset of the bleeding. Then a second course is given. Equally good results have been obtained by the use of diethyl stilbestrol tablets 0.5 mg. twice daily for two weeks followed by 0.5 milligrams daily given concurrently with 10 milligrams pregneninolone daily the third week in twenty cases treated in 1942 at the University Health Service. Those treated during this past year are not included because they have not been followed for a long enough period of time.

The immediate control of severe bleeding may be the real problem to the clinician. Rest in bed and oxytocics may control the hemorrhage. Posterior pituitary extract or ergonovine may be used as a temporary control of the bleeding. A curettage should be performed if the bleeding continues, with packing of the uterus and vagina if necessary. Blood transfusions should be given in cases of severe anemia. Good results have been reported from using pregnant donors giving the patient both blood replacement and possibly the benefit from the gonadotropic hormone contained in the blood. Lactating women have also been used, giving 15 to 40 c.c. of their blood intramuscularly (Greenblatt and Torpin[10]).

Moccasin snake venom has also been reported to have given good results. In the two cases in which it was used by the author it did not affect the bleeding. Vitamin K may be given although its use is empirical.

General health measures are important in preventing recurrences of abnormal bleeding. A well-balanced diet should be prescribed including sufficient vitamins, liver and iron. Insulin therapy in the undernourished should not be overlooked. Mental and physical rest is also important in this developmental period. Psychotherapy may account for many of the symptomatic results from various treatments. The change in amount and duration of bleeding and regulation of the cycles were frequently noted in a group of college women treated with placebo in a controlled study of dysmenorrhea by the author.[1]

Low-dosage irradiation of the pituitary or ovary must be used with great caution in the adolescent. Drips[6] reported good results in a small number of patients. Irradiation may be a method of choice in intractable cases. Intrauterine radium must also be used with great caution. Keene and Payne state that the initial dose should not exceed 200 milligram-hours in women under twenty years of age, with an additional 100 mg. if further treatment is necessary. Hysterectomy may be considered in preference to irradiation or radium in intractable cases.

In nonmenstrual bleeding not under ovarian control the underlying pathology determines the specific therapy.

Conclusions

1. Of 7,634 women entering the University of Minnesota from 1938 to 1942, 22 per cent complained of irregular menstruation, 225 of these complaining of profuse bleeding.

2. During these four years 2,302 students have consulted the gynecological department of the Student Health Service, 630 of whom came because of irregular bleeding.

3. A modified Schroeder classification has been used in our study of uterine bleeding.

4. Hormone assays have been done on the urine in normal cases, and on patients with irregular shedding, anovulatory bleeding, cystic glandular hyperplasia, and oligomenorrhea.

5. The effects of the administration of the various hormones on the excretion products in the urine have been studied.

6. Hormones should be administered to patients with abnormal bleeding in the adolescent with caution and not until a careful physical examination including laboratory tests have been made to determine the cause of the bleeding.

7. The author's experience in treating abnormal bleeding in young college women is briefly reviewed.

8. Abnormal bleeding is frequently self-limited and results due to spontaneous remissions or other fortuitous factors must be kept in mind before one judges the efficacy of any therapy.

9. Physicians should not become impatient with the slow progress made in the study of the hormones.

10. Continued clinical application of the increasing knowledge of endocrine function will aid in the treatment of abnormal uterine bleeding.

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HEMORRHAGE IN PREGNANCY

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STATISTICS on maternal mortality still show a high percentage of deaths due to hemorrhage, and it has been emphasized that in this State,[18] the decrease in the number of maternal deaths due to sepsis and toxemia has not been paralleled by a similar decrease in deaths due to hemorrhage. In the country as a whole, there has been a marked decrease in the number of maternal deaths due to hemorrhage since the more general availablility of blood transfusion owing to establishment of blood banks and the discovery of new blood substitutes as well as the use of ergonovine in the third stage of labor. Attention has been drawn, however, to the fact that many deaths listed as due to hemorrhage are in reality due to shock[41] and that the reduced mortality rate mentioned above concerns cases of hemorrhage per se. It has also been indicated that in spite of the greater facilities for blood replacement, these are not always promptly utilized. Precious time is frequently lost owing to lack of precision in diagnosis or to confusion because of lack of standardization of techniques of treatment. By routine recording of the specific gravity of the blood shortly before delivery and of the amount of blood lost in each case, with the use of soft tissue roentgenograms and the records of the results of various therapeutic procedures in given types of hemorrhage, it should be possible to achieve more accurate diagnosis, an earlier evalution of new methods of treatment and their more rapid and general application.

Read at the annual meeting of the Minnesota State Medical Association, Minneapolis, May 18, 1943.

A study of the records from the maternal department of the St. Cloud Hospital of St. Cloud, Minnesota, over a period of twenty years, shows that in 1922 there were 172 deliveries and in 1942, 925 deliveries. During the twenty-year period, there were 8,321 deliveries, with thirty-five maternal deaths from all causes, of which eleven were due to hemorrhage (two cases of shock, four cases of placenta previa, four cases of postpartum hemorrhage and one case of uterine rupture). The amount of blood loss in a series of 407 deliveries at this hospital is shown in Table I.

TABLE I. BLOOD LOST POSTPARTUM

Age	60 to 200 c.c.	200 to 400 c.c.	400 to 600 c.c.	600 to 800 c.c.	800 to 1000 c.c.	1000 to 1200 c.c.	1200 to 1400 c.c.	1400 to 1600 c.c.	1600 to 1800 c.c.	1800 to 2000 c.c.	2000 to 2100 c.c.
Grav. I											
16—25	24	28	10	6	0	0	0	1	0	1	0
25—30	9	19	7	3	2	1	0	1	0	0	0
30—35	15	13	4	3	1	1	0	0	0	0	0
35—40	5	2	2	2	0	0	0	0	0	0	0
40—46	2	2	0	0	1	0	0	0	0	0	0
Total	55	64	23	14	4	2	0	2	0	1	0
Multi-grav.											
16—25	8	10	4	2	2	1	0	0	1	0	0
25—30	32	35	7	5	4	3	0	3	0	0	0
30—35	33	23	10	3	0	0	0	0	1	0	1
35—40	14	10	5	2	0	2	0	2	0	0	0
40—46	7	5	3	2	0	0	1	0	1	0	0
Total	94	83	29	14	6	6	1	5	3	0	1

The causes of excessive postpartum bleeding are: (1) low placental implantation; (2) uterine inertia; (3) relaxation of uterus due to too deep surgical anesthetic in repairing episiotomies; (4) too hasty removal of the placenta.

None of the above patients who lost more than 600 c.c. of blood had any ill effects. Each recovered within ten days and was able to leave the hospital.

The causes of hemorrhage are different in the three trimesters of pregnancy. When bleeding occurs during the first trimester, the most common cause is abortion or ectopic pregnancy. Other conditions which may cause hemorrhage during early pregnancy include retroversion, multiple pregnancy, syphilis, toxemia, subacute salpingitis, decidual endometritis, uterine myomas and pulmonary tuberculosis. Also deep cervical tears,[15] hydatidiform mole,[9] congenital malformations of the uterus, cervical erosions, varicas,[22] or carcinoma and vaginal, labial or extragenital lesions may be responsible.

Various methods have contributed to greater accuracy in the diagnosis of these conditions, such as observance of the time of onset of bleeding, the degree of hemorrhage, the condition of the blood and the presence or absence and type of pain. Decidual biopsy[38] has been recommended by some writers as a means of ascertaining whether abortion is inevitable. Also the Achheim-Zondek test for hydatidiform mole[9] and the roentgen ray diagnosis of tubal pregnancy, placental insertion and detection of monster feti have proved valuable.

In differentiating between abortion and ectopic pregnancy,[37] it is emphasized that the former usually occurs between the eighth to twelfth week and the latter from the fourth to sixth week. The bleeding in the former is usually profuse and bright red in color, and the pains rhythmical, whereas in the latter, hemorrhage is less abundant, the blood is darker containing decidual rests, and the pain sharper. Ectopic pregnancy has to be differentiated from acute appendicitis, acute adnexitis, follicular rupture, ruptured corpus luteum, ruptured pyosalpinx, cystadenoma of the ovary with twisted pedicle, hemorrhagic pancreatitis, and perforation of the bladder.

Abortion is the most common cause of hemorrhage in early pregnancy, and may be induced or spontaneous, threatened, habitual, or inevitable. The abortion may be complete or incomplete, or missed with retention of the fetus. Each of these conditions has its own special indications for therapy. Prophylactic measures in cases of threatened and habitual abortion include administration of vitamin E[42] and C,[27] progesterone, thyroid and opiates. Opinions vary as to the use of morphine, as the resulting relaxation of the cervix and stimulation may transform a threatened abortion into inevitable abortion. Oral prenone has been recommended in doses of 10 mg. twice daily until symptoms subside, and then twice weekly for several months. If this fails,

digital exploration of the uterus and evacuation of its contents may be necessary.

In cases of illegal abortion associated with bleeding, any placental tissue in sight may be removed with sponge forceps. Curettage is not a safe procedure in the presence of frank sepsis. In fact, it is nearly always fatal. Oxytocics to stimulate uterine contractions, may control bleeding. Locally nothing should be done, besides an ice bag to the lower abdomen until the septic condition improves. The alcohol drain in these cases, as suggested many years ago by Dr. E. J. Ill, is most valuable.

In cases of incomplete abortion with hemorrhage, the aim of treatment is to aid in delivery of the fetal products from the uterus. This may be achieved by administration of pituitrin, ergot or both intramuscularly with careful attention to immediate availability of emergency measures to combat hemorrhage.

In complete abortion the bleeding as a rule subsides once the fetal products have been passed and involution can be accelerated by administration of oxytocics.

In inevitable abortion the symptoms differ only in degree from those of threatened abortion. The hemorrhage and pains are more severe and the bleeding may become profuse before the membranes are expelled. If the hemorrhage is profuse, emptying of the uterus is indicated. Expulsion may be hastened by administration of pituitrin or ergot. If bleeding is not excessive one may proceed expectantly awaiting expulsion.

In missed abortion, the fetus dies in utero and the membranes are retained with atrophic changes. The fetus may be retained for months and is eventually expelled in its dried state. Before expulsion, intermittent bleeding occurs and may last for several weeks. This is followed by profuse bleeding, pain and expulsion. Some authorities prefer to await expulsion, others administer estrogen[19] to expedite matters.

It has been estimated that 98 per cent of ectopic pregnancies occur in the tubes. If the fertilized ovum happens to be near the outer end of the tube, the developing embryo will cause enlargement of the lumen of the tube, with extrusion of the embryo and membranes into the peritoneal cavity, which is accompanied with more or less bleeding. Hemorrhage may be moderate and repeated or profuse, leading to severe shock. Rupture of extrauterine pregnancy in the isthmial portion of the tube is manifested by sudden, excruciating pain, vomiting, blanching, rapid weak pulse and collapse (one such case was seen in the writer's office). The treatment is laparotomy, but only after shock has been cared for. Simultaneous blood transfusion during operation, or reinfusion of the patient's own blood, has yielded good results in many cases.

Extrauterine pregnancy within the uterine wall may break into the uterine cavity, with profuse vaginal bleeding and go on to term due to sufficient placental attachment to the uterine wall. The only symptom is the bleeding which occurs at the time of expulsion. One case so diagnosed prior to an estimated blood loss of 500 c.c. went on to term.

In multiple pregnancy, hemorrhage occurs when the placental attachment is low, marginal, or over any portion of the internal os. The bleeding may be slight or profuse and with or without pain.

In retroflexion, the only symptom may be mild or even profuse bleeding over a period of several days (up to eighteen days). After replacement of the uterus, the bleeding may stop within forty-eight to seventy-two hours without interruption of pregnancy.

Bleeding during the second trimester is infrequent, and when it occurs, may be due to the same causes as in the first trimester, to hydatidiform mole or placenta circumvallata. In hydatidiform mole,[9] the bleeding may last for several weeks. The bleeding is intermittent and the uterus enlarges symmetrically, followed later by spontaneous expulsion of the mole.

In circumvallate placenta, the only symptom is the loss of blood which may last for several weeks before labor is established.

Bleeding is infrequent during the third trimester of pregnancy,[43,46,48] and when it occurs, may be due to extragenital or genital lesions. Among the former may be mentioned circulatory disturbances, nephritis, and the blood dyscrasias.[12,40] The genital lesions include extrauterine pathology such as tears,[15] hematoma,[45] necrosis and varicosities.[22] The intrauterine conditions are placenta previa, premature separation of a normally implanted placenta and tumors.

In cases of bleeding in the third trimester, no time should be lost in getting the patient to the

hospital with a vaginal pack before transportation only in extreme cases. No vaginal examination should be made before the patient arrives at the hospital and then only when full preparations for transfusion, delivery or operation are in order. Even gentle manipulation of the placenta may precipitate hemorrhage. Soft tissue roentgen examination may aid in ruling out placenta previa. A sterile vaginal examination and roentgenography may spare the patient needless termination of pregnancy.

Cystography will aid in differentiating between cases of partial and total placenta previa. The absence of pain should rouse suspicion of placenta previa, but Wallace[46] has recently emphasized the fact that pain is present in a large number of these cases. If placenta previa becomes evident during the second stage of labor, conservative expectant treatment is justifiable. Treatment for shock should always be administered before delivering the patient. The decision as to whether vaginal or abdominal delivery shall be employed will depend upon the degree of hemorrhage, the type of placental insertion, viability of the fetus, degree of cervical dilatation, the time of onset and the parity of the patient.

Surgical treatment is said to give best results in primiparae. In multiparae simple rupture of the membranes is the treatment of choice. In a recent series of 2,000 cases analyzed by Ramirez Olivella,[29] with an incidence of one in every 250 deliveries, classical cesarean section was found to give the poorest results. The fetal mortality rate was in inverse proportion to the age of gestation, and in direct proportion to the site of insertion, i.e., the lower the insertion the higher the mortality. Most of the maternal deaths were due to hemorrhage or infection.

Davis,[13,14] of Nebraska, obtained best results by simple rupture of the membranes and second best by use of Willets scalp traction. Cesarean section offers best prospects for a living child. Pre-operative roentgen examination may be of aid in determining the type of section. Davis found results of Braxton Hicks' version, uterine pack, vaginal cesarean section and manual dilatation of the cervix to be poor.

It is well to keep in mind that the more central the location of the placenta, the earlier does bleeding take place. Spontaneous separation of the placenta with or without external bleeding causes early pain which subsides if the blood escapes from the uterus. In concealed hemorrhage, the pain is constant and severe, with board-like hardness of the abdomen, absence of fetal heart sounds in complete separation or in the presence of a hematoma under the placenta. Roentgen examination may reveal an outline in a low implanted placenta.

Intrapartum Hemorrhage

Intrapartum hemorrhage is most often caused by partial or complete spontaneous separation of either a normally implanted placenta, placental attachment to the upper portion of the lower uterine segment or from rupture of a vessel in a velementous attachment of the cord. This type of hemorrhage is usually concealed until the head slips out of the cervix or after the delivery of the body. The low attached placenta causes the greatest amount of bleeding and is very difficult to deliver, as it does not readily separate due to failure of good contraction and retraction of the lower uterine segment. The bleeding is profuse and cannot be controlled by grasping the uterus, giving ergot, pituitrin or both early, until there is complete separation of the placenta. My practice is to give pituitrin (0.13 to 0.19) shortly before the delivery of the head and ergotrate 0.0003 immediately after the head is delivered. This, too, has failed to give the desired results in many cases. No difficulty has ever been encountered in delivering the placenta due to medication.

In premature separation of the placenta, Olivella[29] found an incidence of 0.14 per cent. This condition occurs most frequently in multipara and exclusively in the last three months of pregnancy. It is toxic in origin with increase in uterine tonus, painful hemorrhage, albuminuria and cylindruria. Amputation of the uterus is unnecessary. Conservative treatment is recommended.

In an analysis of ninety cases, Boulware[5] states that multiparity adversely affects prognosis, while fetus prognosis is unfavorable regardless of the severity of the disease or the type of therapy. The most important phase of treatment is blood replacement. Manual dilatation of the cervix should be avoided. Mild cases may receive expectant treatment. Repeated transfusions may be required. If the prospects for normal delivery seem poor, cesarean section must be done. It

is important to treat shock by blood replacement before delivery.

At the Chicago Lying-In Hospital, these cases are given intravenous hypertonic glucose and antishock treatment. Simple rupture of the membranes or simple low forceps are indicated. Cesarean section and accouchement forcé should be avoided if possible. If the patient is in labor she should be delivered vaginally; if not, by cesarean section, but with transfusion during operation. Whereas there has been a clear trend for experts to limit the use of cesarean section in the treatment of premature separation of the placenta, Miller[26] claims that this operation should be extended to all cases in which ideal conditions obtain. He states that with a proper selection of cases, there is no increase in mortality, no serious limitation to childbirth, and the fetal mortality is lower. Also Rauramo[35] reports better results with active treatment.

If the condition should prove to be a genuine uteroplacental apoplexy in which the uterus no longer responds with contraction to pituitrin, hysterectomy is indicated. It has been emphasized that in these cases removal of the uterus does not remove the underlying pathology which is believed to be a toxemia. The apoplexy may extend to organs other than the uterus.

Most cases of retroplacental hemorrhage will respond to obstetrical medical treatment. Observation during the postpartum period is of great importance. If hemorrhage persists laparotomy is indicated.

Rupture of the uterus occurs more frequently than supposed.[1,6,33,47] It may be due to scars from previous cesarean sections, from curettage, myomectomy, plastic operations on the uterus, manual removal of the placenta, fibroids, congenital malformations of the uterus, penetration of hydatidiform mole, over-distention due to multiple pregnancy, polyhydramnios, especially in the presence of scar, premature separation of the placenta, ventrofixation with extreme sacculation of the posterior lower uterine segment, version, internal podalic version, uncorrected breach presentation or pituitrin. Immediate revision of the uterus is in order after version. Both maternal and infant mortality rates are high. Recently a fatal case was reported of spontaneous rupture in the fourth month of pregnancy with no vaginal but intraperitoneal hemorrhage. Secondary rupture[33] is rare and occurs more frequently after the classical than after low section. Laparotomy may reveal conditions permitting suture of the scar, or necessitating removal of the uterus. Blood transfusion is recommended.

In cases of ruptured varix,[22] the mortality rate is very high if the patient is in labor. Only one case has been diagnosed prior to operation or autopsy. The symptoms are those of acute internal hemorrhage. Immediate transfusion and operation are indicated. Löhnberg reports a patient saved by hysterectomy. Six of the eleven cases reported in the literature were successfully treated by cesarean section.

Of course, lacerations due to trauma of the perineum, vagina and cervix will cause profuse bleeding and require surgical treatment.

Postpartum Hemorhage[10,13,16,21,24,25]

The causes[28,32,34,44] of postpartum hemorrhage include instrumental delivery, uterine atony, injuries sustained during the terminal portion of the first stage and during the second stage of labor, exhaustion or over-distention of the uterus, ill-judged sedation, too deep anesthesia, prolonged labor, premature separation of the placenta or retained placenta. A large baby, twins, multiple pregnancy or polyhydramnios may cause overdistention of the uterus. Relaxation of the uterus may occur as late as three weeks postpartum and one case was seen in which bleeding persisted for three months due to placental retention.

According to Conn,[8] a blood loss of more than 0.5 per cent of bodyweight constitutes postpartum hemorrhage. Shock is rare with a loss of 500 c.c. of blood unless there are other complications. Also the specific gravity of the blood may be taken as an index of the blood loss to be expected in the third stage of labor.

In prevention of postpartum hemorrhage one of the most important items is proper management of the third stage of labor. Conn is of the opinion that if the uterus is pushed into the pelvis, it should be elevated immediately after the placenta has been expelled, by combined vaginal and abdominal pressure if necessary. He injects ergonovine (0.2 mg. ergotrate) immediately after the birth of the baby and has observed no marked tendency of the cervix to contract or toward incarceration of the placenta as a result thereof. Placental separation is speeded up by this pro-

cedure. Davis[13,14] states that intravenous injection of ergonovine in the third stage of labor perfects the normal mechanism and the increased tonicity guards against further blood loss following placental separation. The tonicity is maintained for several hours thus preventing postpartum hemorrhage. He experienced a few cases of incarceration of the placenta but these could easily be treated at the hospital. Davis gives 0.2 mg. of ergotrate intravenously after the head is delivered and the anterior shoulder exposed. After twenty to thirty seconds, the baby can be delivered. The uterus must be contracted before expression of the placenta begins. The entire uterus must not be pushed down into the pelvis because such a procedure leads to profuse hemorrhage. Davis states that exerting pressure with the fingers of the left hand just above the symphysis, while the right hand pushes down on the uterus may help to avoid such a result. Slight traction on the cord will deliver the placenta when it is presenting at the introitus.

Hunt[18] emphasizes the importance of simple measures. For cases in which simple expression does not suffice to deliver the partially separated placenta, he uses a Crede procedure. If this fails the placenta is removed manually and the uterus packed with iodoform gauze. For hemorrhage immediately following the third stage of labor, the uterus is massaged. Bimanual compression is applied and ergonovine administered. If this fails, the uterus and vagina are packed and if hemorrhage still persists tamponade is repeated, after which in case of continued bleeding hysterectomy is performed. Manual removal of the placenta should not be delayed until the patient is exhausted from loss of blood. Adequate blood replacement is indicated. Hunt emphasizes the non-obstetric methods for control of postpartum hemorrhage including blood replacement by transfusion of blood, plasma or other blood substitutes such as placental blood, saline or glucose solutions, and acacia solutions. He discusses vasoconstrictors, the importance of preventing shock levels of blood pressure for more than twenty minutes, of using positive pressure for giving solutions in states of shock and the dangers of administering saline solutions in toxemia and glucose solutions in diabetes. He urges that efforts be directed toward early control of hemorrhage, as even the life-saving transfusion of blood carries with it possible risk to the patient.

It has been stated that postpartum hemorrhage has been observed with much less frequency after the introduction of the use of pituitrin in the third stage of labor. McGee[25] has been unable to confirm any acceleration of involution following administration of ergotocin and has observed several cases with severe reactions. The latter may occur also after administration of pituitrin. Navratil[28] uses hot vaginal and uterine irrigations if bleeding persists after evacuation and massage of the uterus. In cases of severe atony, aortic compression for ten to fifteen minutes at a point not higher than the level of the third lumbar vertebra may prove useful.

Briscoe and Wilson[7] have obtained satisfactory results by injection of 1 c.c. of pituitrin directly transcervical into the lower uterine segment (firm contractions may thus be produced in eighty-four seconds). The substance is injected into the wall of the uterus to a depth of 2-3 cm. in the anterior fornix. Hoyt[17] describes intra-abdominal injection of pituitrin into the uterine wall in emergency cases.[36]

The necessity for distinction between shock due to hemorrhage and genuine shock both as regards diagnosis and treatment is repeatedly indicated.[39] Shock often persists after hemorrhage has ceased and is not always in proportion to the degree of hemorrhage. The first stages of shock may be masked by the acute stimulation of the operation, ergot, pituitrin, analeptics and cardiacs. Two hours later alarming shock may develop, lasting from six to twelve hours. The pulse is misleading but the facies and warmth of the extremities indicate a good prognosis in cases in which the pulse is poor. Schaefer[39] recommends blood transfusion followed by continuous drop infusions for the treatment of shock. He states that it is the continuous and prolonged administration rather than the quantity of blood transfused that is the determining factor. Schaefer uses the central and peripheral drugs alternately, giving veritol after preliminary blood transfusion and drop infusion. He also warns against rapid succession of individual doses.

With the large number of articles appearing in medical journals on the control of postpartum hemorrhage, including advances in the technique of treatment and diagnosis as well as in the

knowledge of the underlying causes of the hemorrhagic condition itself and of reactions to the various drugs and procedures used, it is to be hoped that obstetricians may profit by the example of speeded up organization and application of findings set by the War Program.

A brief review of suggestions obtained from the literature seem to hold some promise for a further reduction of mortality rates in this field. To begin with let us stress the need for full use of facilities for prompt blood replacement, whether by autoinfusion,[11,31] blood transfusion or infusion of blood substitutes such as plasma, placental blood,[3] saline, glucose or acacia solutions with due regard for proper timing and contra-indications presented by various diseases as well as careful attention to proper matching of bloods and vigilance for detection of the recently emphasized Rh-factor.[18] It has been emphasized that saline infusions or plasma may suffice to tide the patient over until blood transfusion is possible. Likewise it has been stressed that it is the prolonged and continuous support[39] of the circulation by drop infusions following transfusion that are a determining factor rather than the actual amount of blood injected. Frequently massive fatal hemorrhage is preceded by minor hemorrhage, and careful utilization of the time interval between the two in adding to the patient's blood reserve may save life.

Routine roentgen studies and whenever possible permanent records in the form of lantern slide libraries should be encouraged, as also routine measurements of the specific gravity of the blood shortly before delivery and of the amount of blood lost. Careful differential diagnosis and differential treatment for genuine shock and shock due to hemorrhage must be indicated. Records should also be kept of the results of various forms of treatment for the different types of hemorrhage to permit correct evalution and more thorough standardization of techniques without sacrifice of individualization.

It is claimed that the tendency to bleed is greater in anemic blondes and red haired individuals than in brunettes. In spite of an alleged greater tolerance for blood loss in parturients the reaction to blood loss varies individually. Shortening the third stage of labor by means of pituitrin and ergonovine administered in the placental stage seems to diminish the tendency to postpartum hemorrhage.

The sulfonamides[20] have been indicated in a few instances as prophylactics in cases of feared infection, and for uterine packs.[2] Several articles indicate the need for more careful technique in packing of the uterus.[4] The use of analgesics should be limited to the earlier stages of labor so that they may not interfere with proper contraction of the uterus.

It has been emphasized that many deaths are due to application of usual methods for combating postpartum hemorrhage in cases of uterine apoplexy, in which the uterus can no longer respond to stimulation. Here operative treatment is indicated if the apoplexy extends beyond the genital system, as it frequently does because the underlying pathology is probably a toxemia. The latter must be prevented by proper prophylactic hygienic and dietetic treatment during pregnancy. Among the general measures and substances used prophylactically and therapeutically in obstetric hemorrhage may also be mentioned ice-bags, the vitamins E, C, and K and the oral or intra-uterine administration of gelatine, calcium and quinine.

Case Reports

Case 1.—Mrs. C. J. S., aged twenty-six, gravida 3, had her last menstruation April 20, 1940. When she was three and a half months pregnant, there was a spontaneous gush of fluid with bleeding, which lasted for three days (estimated blood loss 300 c.c.) without pain. The patient remained in bed for a week, after which the fluid loss and bleeding ceased and she was able to resume her household duties. She carried to term, delivering a full term, normal male child. The amniotic fluid obtained at delivery amounted to about 45 c.c. The placenta was expressed (Crede), Schultze's mechanism, measuring about 18 x 20 cm. and weighing 610 gms. The cord measured 60 cm. The attachment was lateral. The amount of blood lost was 900 c.c. The area where the rupture occurred in the membranes was sealed by fibrin. The fibrin clot measured 6 x 7 cm.

Diagnosis: Hemorrhage due to spontaneous rupture of the membranes, carried to term.

Case 2.—Mrs. F. J. R., aged thirty-seven, gravida 8, was admitted to the hospital December 5, 1938, because of bleeding which started one week previous. She had her last menstrual period July 8, 1938, and her confinement was calculated for April 15, 1939. Her clothes suddenly became soiled. On examination she found the fluid mixed with blood. The escape of fluid and blood recurred every forty-eight hours and at night. She suffered some abdominal discomfort after the initial showing, which subsided when the fluid passed.

Physical examination was negative except for enlargement of the abdomen to the size of a five months' gestation. There was a vaginal discharge mixed with blood. Upon x-ray of the abdomen, the placenta could not be outlined. The breech was presenting. Red and she felt well. In April, 1938, she had a showing which lasted for four days. On October 16, she started to bleed, and this continued for two weeks. On November 26, there was moderate bleeding, followed next day by profuse hemorrhage which had to be controlled

Fig. 1. *(left)* Photograph of the placenta and membranes (Case 1), showing site of spontaneous rupture of the membranes which was closed by deposit of fibrin.
Fig. 2. *(right)* Photograph of placenta (Case 2), showing the collar-like attachment of the membranes about the origin of the cord.

blood count 3,500,000; white blood count 11,000; hemoglobin 62 per cent.

The placental souffle could be heard with the stethoscope on the right side in the lower uterine segment. After four days in the hospital, the patient was discharged.

On February 6, 1939 the patient was readmitted to the hospital in labor. During her stay at home, small amounts of blood-stained fluid had escaped daily. Two days prior to her admission, she felt much better but the fetal movements were absent. She expelled two old blood clots prior to spontaneous delivery of a male child. The cord made four complete turns about the child's neck, with the cord slipped through the last loop, making a single tie about the neck. The child was cyanosed, and failed to respond to treatment, expiring after forty minutes. The placenta was expressed. It measured 13 x 15 cm. and weighed 450 gms. The cord measured 60 cm. Attachment was central. The membranes were reduced to a small, collar-like area about the attachment of the cord from premature separation about the periphery.

Diagnosis: Circumvallate placenta.

Case 3.—Mrs. F. B., aged forty-four, gravida 7, had her last menstrual period December 26, 1937, followed by severe headache, inability to sleep, and vomiting which was relieved by 10 per cent glucose intravenously, and repeated daily for four days. The patient improved. The headache subsided and she was able to sleep and retained her food. The general physical findings were negative, except for the enlargement of the uterus. Her progress was satisfactory up to five months when her abdomen became soft and began to decrease in size, with loss of appetite and headaches lasting about two weeks. Her appetite then improved by a vaginal pack. As the hemorrhage recurred the following day, the uterus and vagina were repacked, and she was given 1 c.c. each of ergotrate and pituitrin in 1000 c.c. normal saline, by hypodermoclysis. Blood pressure 94/68; red blood count 3,480,000; white blood count 13,000; hemoglobin 66 per cent. The patient was typed for transfusion. On November 29 the pack was removed and replaced. Severe contractions followed with expulsion of the placenta. The placenta was oval in shape and the membranes were intact. It measured 8 x 10 cm., 2.5 cm. thick and weighed 130 gms. The membranes were opened but there was no evidence of a fetus. The blood loss could not be estimated, because the blood was absorbed by pads. Recovery was uneventful. Blood picture before discharge showed red blood count 3,400,000; white blood count 11,800 and hemoglobin 68 per cent.

Diagnosis: Missed abortion.

In a study of the records in the St. Cloud Hospital for the past two years, in over 1,500 deliveries where ergotrate was given only four manual deliveries of the placenta were recorded. The records do not state whether the removal was due to incarceration or to the use of ergotrate.

In a review of 500 of my own cases I find that the placenta was removed three times: once where the placenta was attached to the lower uterine segment and twice where there was a tubal pregnancy of the interstitial portion of the tube. The history shows there was early bleeding which subsided without interruption of preg-

nancy. On attempted removal, the placenta was located in the horn of the uterus like a pocket in a billiard table with a portion of the placenta attached to the posterior uterine wall.

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HEMORRHAGE IN THE MENOPAUSE

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IN the latter part of the menstrual life of women, variations of the menstrual flow are to be expected as a consequence of failure of the function of the ovaries. The menstrual interval may be shortened or lengthened, and the amount of flow increased or decreased. Certain benign lesions may cause similar changes. Occasionally, such changes may be encountered in the presence of a malignant lesion. More frequently, the latter gives evidence of its presence by bleeding between the regular flow, intermenstrual spotting or metrorrhagia. However, the type of atypical bleeding from the genital tract is not sufficiently characteristic to allow a diagnosis of the cause to be made without a careful examination of the pelvic viscera. As will be indicated, this examination must proceed in a more or less orderly

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sequence until satisfactory cause or causes have been established. For example, it should be remembered that fibroids and cancer, or senile vaginitis and cancer may co-exist.

The minutiæ of bimanual examination are familiar to all physicians. After the bladder and rectum have been emptied, the patient should be placed on the examining table in as comfortable a position as possible. Gentleness on the part of the physician and complete relaxation on the part of the patient will contribute greatly to an accurate investigation. Perhaps examination through the rectum is not performed as frequently as it should be. By this means the bases of the broad ligaments, the cul-de-sac of Douglas and the regions of the tubes and ovaries often can be felt more satisfactorily than through the vagina. Examination of the patient in the knee chest position gives valuable information. The vaginal walls and uterine cervix should always be inspected through a speculum. The ordinary bivalve speculum is usually satisfactory; however, the tubular, or Ferguson, speculum has certain advantages. When the largest size that can be introduced is employed, it permits complete visualization of the cervix as well as of the anterior, posterior and lateral vaginal fornices. When the instrument is withdrawn slowly, the vaginal wall can be completely visualized as it rolls over the edge of the speculum. If any portion is suggestive of a malignant lesion, a specimen should be removed for microscopic examination. Schiller's test, made by applying compound tincture of iodine to lesions, and the use of the colposcope are frequently mentioned as part of the examination. I have little faith in the former and believe that the only safe procedure is microscopic examination. It is true that the application of a solution of iodine to the cervix may afford a better notion as to the region from which a specimen should be removed for microscopic study. Malignant lesions do not stain with the solution owing to a lack of glycogen, which is present in normal tissue. Such a failure of staining may be noted in the presence of cervicitis without malignant change. When tissue is obtained for microscopic study, several specimens should be removed. The depth of the specimen should include all the layers of tissue under suspicion. One must be trained in the use of the colposcope. A good one is expensive and, while it may aid in selecting a region for study, it is not possible to make a definite diagnosis with this instrument without microscopic examination.

If no adequate cause of the bleeding is observed in the vagina or on the cervix, the cervical canal should be probed. If bleeding occurs after this procedure, a specimen for biopsy should be obtained from this region.

If atypical bleeding occurs from the genital tract and if no cause for the bleeding can be found in the vagina or cervix, one must perform a dilatation and curettage for diagnostic purposes. This should be done with the patient under anesthesia and the uterine cavity should be curetted thoroughly. The cannula curette used for removal of tissue in the office for determination of the histologic structure of the endometrium has no place in the diagnosis of pathologic conditions of the endometrium. All scrapings should be examined under the microscope by a competent pathologist. Preferably, this examination should be made, by the frozen section technique, while the patient is under anesthesia, in order that further treatment, if indicated, may be carried out immediately. One is likely to consider diagnostic dilatation and curettage as a final and complete method of excluding carcinoma of the body of the uterus if negative results are obtained. In the main, this is true; however, if intermenstrual spotting of blood has occurred, if postmenopausal bleeding is present, or if the uterus is somewhat enlarged and soft, it may be good judgment to remove the uterus, even if microscopic examination does not reveal evidence of malignancy. Under these circumstances, one not infrequently will find an intra-uterine polyp or small carcinoma that has been missed with the curette. Postmenopausal bleeding occasionally is encountered in cases in which the pathologist finds evidence of proliferation of the endometrium in the scrapings obtained by curettage. This should lead to the suspicion of a granulosa cell or a theca cell tumor of the ovary.

In the majority of cases in which atypical bleeding occurs from the genital tract in the latter years of the menstrual life of women, the bleeding will be due to benign lesions. These are: prolapse of the urethral mucosa with meatitis, urethral caruncles, vaginal infections due to Trichomonas and Saccharomyces, senile changes in the vaginal mucosa, cervicitis with ectropion, cervical polyps, benign proliferation of the endometrium, uterine polyps, submucous fibroids, re-

tained secundines, and an atrophic endometrium.

Prolapse or eversion of the urethral mucosa or a urethral caruncle associated with meatitis occasionally is the only reason for bloody spotting. A caruncle should be removed by fulguration. The prolapsed mucosa usually is best left alone if the meatitis can be corrected by local treatment.

Infections of the vagina need not be considered in detail. Their diagnosis and proper treatment have been considered frequently and extensively. It may be mentioned, however, that when these infections occur in a senile vaginal mucous membrane their treatment may be more difficult because of the poor condition of the infected tissue. Frequently, sufficient estrogenic substance to afford rehabilitation of the mucous membrane of the vagina will facilitate the treatment usually employed. Indeed, in the case of infection by Trichomonas, this may be sufficient treatment.

One not infrequently sees bloody spotting due solely to the senile changes in the vaginal mucosa. The picture is quite characteristic. The tissue is thin and pale and it may be diffusely reddened. Petechial regions are numerous and slight trauma with an applicator or the edge of the speculum will produce an oozing of blood. Other sources of bleeding should be kept in mind and excluded. If, after treatment, the bleeding disappears and if subsequent examination of the pelvis gives negative results, the senile changes may be considered the source of the bleeding. The condition may be treated by the oral, local or intramuscular administration of estrogens. I prefer to use stilbestrol in doses of 0.25 or 0.5 mg. given daily for three or four weeks. This will usually control the condition although subsequent courses of treatment may be necessary.

Cervicitis with ectropion and erosion may cause intermenstrual bleeding. The cervix should be adequately visualized and carefully inspected. A specimen of the lesion should be examined microscopically before treatment is instituted. Treatment depends on the extent of the lesion. The ordinary cervicitis can be well treated in the office by a nasal tip cautery. If marked hypertrophy and laceration are present, conization, deep surgical cautery, a Sturmdorf operation or amputation are preferable.

Cervical polyps are usually benign. However, malignant change is encountered just often enough for one to recommend the removal of all cervical polyps, which should be followed by microscopic study. Polyps are liable to be multiple, and careful search of the cervical canal should follow the removal of a polyp. The polyp should be grasped with a forceps. The base of the polyp should be severed with the actual cautery and thoroughly destroyed. Failure to do this leads to recurrence.

One must condemn the practice of the treatment of atypical bleeding from the uterus in the latter half of the menstrual life without diagnosis of the cause. Before the menses have ceased, if careful pelvic examination gives negative results and if the patient may be kept under control, a period of observation is allowed. After cessation of menses, if examination of the pelvis gives negative results, except for an obvious senile vaginal mucosa that is bleeding, a period of observation and treatment of this condition may be permitted. It is very bad medical practice to give a woman "shots" for menstrual irregularities when she has reached the age of the menopause, unless cancer has been excluded. If cancer has been excluded and if radical treatment with radiation or operation is not indicated by the patient's general condition, such as a severe anemia, I can see no harm in a trial of hormonal therapy but do not have much faith in success. In many of the cases, atypical bleeding is caused by failure of function of the ovary owing to senescence of this gland and no amount of stimulation of the ovary will have much chance of producing a beneficial effect. Preparations of male hormone, testosterone propionate, are currently employed and at times are beneficial. It must be borne in mind that masculinizing effects may occur and that these are undesirable from the patient's standpoint. It has been stated that no more than a total of 1,000 mg. should be used. This may be given in doses of 20 to 50 mg. twice a week. An average monthly dose is 300 mg.

Cancer of the uterine cervix frequently announces its presence by an intermenstrual or postmenopausal bloody discharge. Yet all too frequently one sees advanced carcinomas, certainly detectable long before by examination, that have escaped the patient's attention because none of the early signs such as leukorrheal discharge and blood have been noticed. Principally, this condition is treated by a combination of radium and roentgen rays. In a small number of cases in which Stage I lesions are present, a Wertheim

hysterectomy may be performed and radiation employed subsequently.

The treatment of carcinoma of the body of the uterus is fairly well standardized and consists of total abdominal or vaginal hysterectomy. Exception occurs when the lesion has metastasized, a complication which fortunately occurs late in the course of the disease, or when the condition of the patient makes the risk of surgical treatment excessive. Then treatment by radiation may be employed.

The treatment of atypical bleeding from the body of the uterus due to benign conditions such as fibromyomas is less standardized. All physicians probably would agree that a diagnostic dilatation and curettage followed by the intrauterine application of 1,200 to 1,600 mc. hours of radium or a dose of roentgen rays sufficient to obliterate function of the ovaries is good treatment for menorrhagia caused by hyperplastic endometrium or submucous fibroids in a case in which the patient is forty-five to fifty years of age, if the uterus is no larger than one that contains a three-month fetus. The patient is at the end of her period of ovarian activity. Such treatment succeeds in the great majority of cases of this type.

What about the same circumstances in a case in which the woman is thirty-eight years of age? Treatment with radiation precipitates the menopause. It has been shown that further bleeding will occur in 30 to 40 per cent of cases in which women less than forty years of age are treated by the means just mentioned. Bleeding is much more likely to develop subsequently if menopausal symptoms need treatment by the administration of estrogens. It seems to me that hysterectomy with preservation of the ovaries is much more sensible treatment for persistent atypical bleeding from the uterus in this group of cases.

One of the most difficult gynecologic opinions concerns enlargement of the ovary in the case of older women. Cancer of the ovary is most frequently seen between the ages of forty and sixty-five years. It does not announce its presence by bleeding from the uterus in many cases. It is detectable in the early stages only by careful examination of the pelvis. This should lead physicians to educate their patients concerning the value of periodic examinations of the pelvic viscera during this period of life. It has been recommended that this be done yearly or at intervals of six months. I do not believe that significant or satisfactory progress in the early diagnosis of cancer of the pelvic viscera of women will be made until this program is carried out. No amount of education confined to the description of the early symptoms of cancer will accomplish much. Every large and easily diagnosed ovarian tumor was once small. If atypical bleeding from the genital tract occurs during the latter part of menstrual life or if postmenopausal bleeding is associated with significant enlargement of the ovary, exploration of the pelvis is to be considered seriously. A period of observation and repeated examinations may be employed before making this decision. Naturally, one must keep in mind other causes of bleeding, for multiple causes may be present.

Summary

Atypical bleeding from the uterus in the latter part of the menstrual life is a significant symptom. Bleeding that occurs from the genital tract after the cessation of menses is increasingly significant. Every woman who consults a physician because of such symptoms should receive a careful and complete diagnostic survey. One should advocate periodic examinations of the pelvic viscera and not rely solely upon education of the laity concerning the early symptoms of cancer—all too frequently there are none. The treatment of atypical bleeding from the genital tract of women without adequate diagnosis is to be condemned severely.

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Milk is the largest single source of farm cash income and was 15 per cent of all farm income in 1942—larger than cattle or hogs, twice cotton, three times wheat, five times tobacco.

"Americans enjoy the best milk in the world," it is said, "with sanitary safeguards and widespread distribution that make this milk supply an invaluable asset in wartime."

EXPERIENCES WITH THE Rh SUBSTANCE IN TRANSFUSION REACTIONS

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THE knowledge of the Rh substance made available by the studies of Landsteiner, Levine, Wiener, and others,[13,15,16,23] has contributed materially to the identification and prevention of transfusion reactions. These new data should rightfully be received with enthusiasm but at the same time they should not obscure in any way the many lessons previously learned regarding the successful management of blood transfusions. This new information is one more forward step comparable to identification of pyrogen reactions, popularization of citrated blood or development of blood storage.

The Problem in General

The following two cases are briefly summarized to illustrate the general nature of the reactions due to Rh sensitization.

Case 1.—This patient was admitted to the hospital because of a pelvic mass. On exploration, a carcinoma of the ovary was found and resected. During the operation, the patient received a transfusion of 500 c.c. of blood without any reaction. Sixty days later, the patient returned to the hospital for a second transfusion at which time the original donor again offered to give blood. The crossmatching done by the Levine method showed marked agglutination of the prospective donor's cells.

Case 2.—A woman near term was delivered by cesarean section. The obsterical history and indications for the section were not stated by the referring physician. A transfusion of group 0 blood from the husband was given at the time of the surgery. The patient suffered a reaction consisting of chills, fever, jaundice and oliguria. She recovered. The bloods were rechecked and were of compatible groups.

Comment.—These two cases illustrate the general character of the reactions due to Rh sensitization. They occur in spite of the use of group compatible blood and they occur after a prior transfusion or during pregnancy.

The Rh Substance

Human blood contains many specific substances[1,5] of which only a few are of interest to physicians. The A and B substances are well known since they are responsible for the division of bloods into the four blood groups: AB, A, B, O (Moss groups 1, 2, 3 and 4, respectively). The M and N substances are useful in medicolegal problems and very rarely are involved as a cause of transfusion reactions.[12,25] Until recently, the Rh substance had no clinical application and for this reason, like most of the other specific blood substances, was unknown to physicians. Now that the significance of the Rh substance has become apparent, a knowledge of its properties has become important.

No definite direct information is yet available relative to the physical properties of the Rh substance. Presumably, like the A and B substances, it is a carbohydrate fixed to the protein of cells. It is fixed to the red blood cells and as yet has not been identified in any of the other body tissues[14,18] although the evidence indirectly indicates that the Rh substance should be present in other tissues, particularly in the liver. It is not present in commercial serum or plasma[10] (see Case 8). Approximately 85 per cent of individuals possess this Rh substance as a normal constituent of their red blood cells. This group is spoken of as Rh positive. Fifteen per cent of people do not have this substance and this group is known as Rh negative.

Although the general principle of sensitization to the Rh substance is simple, the specific details are very confused. Rh positive individuals already have the substance as part of their blood and are not affected by any further addition of it to their bodies. On the other hand, Rh negative individuals do not have this substance in their blood and if it is introduced into their bodies, they react to it as though it were a foreign protein. After an "incubation" period of variable length, they develop antibodies against the Rh substance.

The Rh substance can be introduced into the Rh negative individual in two ways: by transfusion of Rh positive blood or by a pregnancy in which the fetus is Rh positive. Rh negative patients transfused with Rh positive blood have no reaction from the first transfusion but if they are again transfused with Rh positive blood after

they have formed antibodies, they develop a reaction. An Rh negative woman who bears an Rh positive fetus may become sensitized during the pregnancy and if she is transfused with Rh positive blood she will develop a reaction during the first transfusion.

The Rh positive child born of such a sensitized mother shows the syndrome known as erythroblastosis.[11,16] The maternal antibodies pass through the placenta and combine with the fetal antigen. This union in the fetus produces hemolysis and cellular damage to capillaries, liver and sometimes to the brain. The hemolysis results in jaundice, anemia, hyperplasia of blood forming tissues and immature red cells in the circulating blood. The cellular damage produces edema, petechial hemorrhages and sometimes necrosis of the liver and degeneration of brain cells.

The syndrome is extremely variable. The child may be born prematurely or born dead. If born alive, the child may show distress at birth or may live for several days before clinical symptoms appear. Occasionally, the disease manifests itself only as an anemia which may not be apparent until many days or weeks after birth. This syndrome presenting itself in the child or evident from the past history of the mother is an extremely valuable warning to the clinician that the woman may be Rh negative and sensitized to Rh positive blood. Not all erythroblastosis is due to Rh sensitization so that the occurrence of this syndrome in the infant is not absolute evidence that the mother is Rh negative and sensitized.

The various blood substances, Rh included, are in the broad sense of the word, antigens: that is "when introduced parenterally into an animal, will cause the production of antibodies."[2] However, when introduced into humans, these substances act very irregularly. The A and the B substances are strong antigens and if introduced into the body by intention or by transfusion of incompatible blood consistently stimulate the formation of antibodies.[1,19,26] The M substance will stimulate antibody formation only in very exceptional instances.[12,25] The Rh substance is intermediate in this respect. In some Rh negative individuals, the substance is a powerful antigen and stimulates a high titre of antibodies. In other people, the substance is either nonantigenic or very slightly so.[24,27] The following case illustrates the powerful antigenic property exhibited in some individuals.

Case 3.—This woman had had one normal Rh negative baby. Her next two pregnancies resulted in stillborn, premature infants, both dying of erythroblastosis. She was referred for study by her physician during the third month of her fourth pregnancy. Her blood was group A and Rh negative. The husband was a group 0 and Rh positive. Her serum agglutinated most Rh positive bloods. Her serum was studied at monthly intervals throughout her pregnancy. The anti-Rh titre ranged as high as 1 to 250. The child died soon after birth of the hydroptic type of erythroblastosis. On the sixth postpartum day the anti-Rh titre rose to 1 to 5,000. (This case will be reported in detail elsewhere.)

Considerable evidence can be accumulated to show that the Rh substance is weakly antigenic or not antigenic in some people. A study was made of the Rh status of the mother and child in 200 normal deliveries. Blood smears were made on all the babies. All, with the exception of one premature child, had less than seven nucleated red cells per 100 white cells. The premature child maintained a hemoglobin of over 17 grams. In other words, none of these babies showed any evidence of erythroblastosis. Twenty-six of these mothers were Rh negative and their babies were Rh positive. In sixteen of these mothers, serum was studied between the fifth to the eighth postpartum day. Three of these sixteen showed traces of antibodies and were presumably sensitized to a degree which, however, was too mild to affect the child. Twelve of the twenty-six mothers were multipara and had previously delivered a total of twenty-one normal children. Two had had one miscarriage, each in the early months of pregnancy, and none had had an erythroblastotic child. This group of twenty-six Rh negative women with Rh positive babies could have been inoculated with the Rh substance during their pregnancy. However, none showed evidence that such inoculation, if it had occurred, stimulated any significant antibody formation. Since the manner in which the transfer of antigen from the fetus to the mother is unknown, there exists the possibility that not all of these women had been inoculated.

The general incidence of erythroblastosis likewise indicates that only a few Rh negative women exposed to the Rh substance during pregnancy are affected by the exposure. In the general population the theoretical occurrence of an Rh nega-

tive mother bearing an Rh positive child is about 9 per cent. However, the incidence of erythroblastosis which can be taken as a gauge of the frequency of significant sensitization is probably not over 0.5 per cent. Therefore, roughly, only about one in twenty Rh negative women exposed to the Rh substance during pregnancy react with antibody formation. However, those women who do develop antibodies usually do so with every Rh positive fetus. These circumstances strongly indicate that most Rh negative women are refractive to the Rh substance in the same way as the majority of all humans are refractive to the M or N substance.

Experience gathered from inoculation of Rh negative individuals with Rh positive blood by means of transfusion shows similar results. The following two cases serve to illustrate the poor antigenic power in some individuals.

Case 4.—A young woman injured in an automobile accident was admitted to the hospital in deep shock. She had multiple fractures and a rupture of the urinary bladder. Her blood was group A and Rh negative. In the next three days she received a total of 2,200 c.c. of Rh positive blood. Thirty-two days after the first transfusion, questionable traces of Rh agglutinins were present. On the thirty-sixth day, distinct traces were observed and these were absent on the sixty-fifth day. The donors' Rh positive cells were still present in her circulation.

Case 5.—The patient was admitted to the hospital for care of a duodenal ulcer. Shortly after admission, he developed a massive hemorrhage. The hemoglobin dropped from 16 grams to 6.3 grams. The patient was a group 0 and Rh negative. He was given 2,500 c.c. of Rh positive blood. He remained in the hospital forty-four days after the last transfusion. The Rh positive cells were still present in the patient's circulation at the time of discharge. The serum at one time showed questionable traces of Rh agglutinins but these traces were absent a week later.

Comment.—These two patients each received over 2,000 c.c. of Rh positive blood. These patients were studied for sixty-five and fourty-four days after receiving this inoculation. Neither showed any significant titre of antibodies.

It would appear that only about one in twenty Rh negative women bearing Rh positive infants develop significant antibodies. Also it appears that some individuals even after massive direct inoculation by transfusions do not develop demonstrable antibodies of any degree. One is forced to conclude that the Rh substance is not antigenic to all individuals. In spite of the evidence presented, one must reserve final judgment because this entire phenomenon of Rh sensitization is very complex and more experience is needed before a final conclusion is warranted. The following case suggests that factors still unknown may be involved.

Case 6.—This patient was admitted for repair of a cystocele and rectocele. Her last child was born twenty-three years prior. She had a total of four pregnancies, all ending in the delivery of normal children. There were no abortions, no stillbirths and no evidence of erythroblastotic babies. The patient was a group B and Rh negative. The Rh grouping of the husband was not done. Two of the four children were examined and these two were Rh positive. The patient was given 500 c.c. of Rh positive blood. Five days later, another transfusion was to be given. The Levine cross-matching showed distinct agglutination of cells from all of 10 Rh positive donors. By the fourteenth day after the transfusion, her serum produced marked agglutination of all Rh positive test cells.

Comment.—This Rh negative woman had four normal children. Two of these were examined and both were Rh positive. The exposure to the Rh substance during these pregnancies did not produce enough sensitization to cause erythroblastosis in the children. However, when inoculated with Rh positive blood twenty-three years after her last pregnancy, she promptly developed a marked antibody response. Because all of the processes involved in the phenomenon of Rh sensitization are not known, conclusions should not be arrived at too hastily.

The Rh Antibody

The antibodies formed against the Rh substance are present in the serum. They, like other antibodies, probably are proteins contained in the globulin fraction. It must be assumed that, as in similar antigen-antibody relationships, there are several types of antibodies produced: agglutinins, hemolysins, percipitans, cytotoxins and others. No fractionation of these different antibodies has as yet been accomplished. There is clinical evidence to indicate that there exists a multiplicity of antibodies and this variety of antibodies finds an important application to blood transfusions.

Case 7.—This woman had two prior stillborn children. Autopsy examinations had shown typical erythroblastosis in both. She was referred by her physician at about six weeks of her pregnancy. The examination showed that she was a group B and Rh negative. She

had no Rh antibodies in her serum at this time. The next examination at about ten weeks of pregnancy showed traces of Rh antibodies. She was seen periodically throughout her pregnancy and, after the examination at ten weeks, Rh antibodies could no longer be demonstrated. Shortly before delivery, a trace of antibodies again reappeared. The child at birth had a massive hydrops and died in a few minutes. After delivery, the Rh antibodies rose slightly so that definite traces were demonstrable.

Comment.—This woman, studied throughout her pregnancy, never showed more than traces of agglutinins, however, the child in utero suffered a profound reaction sufficient to induce the most severe form of erythroblastosis. It is obvious that this mother was forming antibodies yet none could be demonstrated by matching her serum against Rh positive red cells. Did the child in utero absorb all the antibodies as fast as they were produced by the mother? If this were true, the mother would have necessarily continued to form such antibodies after the termination of the pregnancy and they would have accumulated and been demonstrable after the separation of the child as in Case 3. The probability is very great that this woman formed hemolysins and cytotoxic antibodies which destroyed the fetus but that she did not form agglutinins. If this woman had been transfused with Rh positive blood, it seems very probable that she would have suffered a reaction even though no agglutinins were demonstrable.

In the laboratory, the tests for compatibility of bloods and for the presence of antibodies consist entirely of demonstration of agglutinins. It must be emphasized that the absence of demonstrable agglutinins does not preclude the absence of other antibodies capable of producing a serious reaction.

Case 8.—This patient was four months pregnant and was explored for an upper abdominal mass. An extremely vascular retro-peritoneal tumor was encountered which bled so profusely that the wound had to be closed over packs. Transfusions were given. The third and fourth transfusions of compatible group A blood caused severe reactions and both had to be discontinued. Blood was referred for study at this time. The patient was a group A and Rh negative. Only a few of the transfused cells persisted in the circulation. No Rh agglutinins could be demonstrated either at this time or on two subsequent examinations. While search was being made for Rh negative donors, an infusion of commercial serum was given without reaction. Plasma from a group A Rh positive individual was prepared and this contained a "few" red cells. Infusion of this fluid caused a reaction. Two more infusions of commercial plasma were given without reaction. By this time, Rh negative donors were found and two transfusions of this blood were given, both without reaction.

Comment.—This patient suffered reactions from Rh positive blood and from plasma contaminated with Rh positive cells. She had no reaction from commercial plasma or from Rh negative blood. Her serum studied over a three-week period after the last transfusion with Rh positive blood showed no Rh agglutinins. It appears clear that in spite of the absence of Rh agglutinins, this patient had other types of Rh antibodies capable of inducing reaction.

Other biologic properties of the Rh antibody have direct clinical application. The Rh agglutinin is slow acting and produces its effect best at body temperature. It is this slow activity and need for incubation which prevents the demonstration of these agglutinins in the usual open-slide method of examination. A special technique has been devised by Levine.[16,17]

The mixture of diagnostic antiserum and the unknown cells is incubated at 37° for one hour. The suspension may be examined at thirty minutes and if definite agglutination is visible, no further incubation is necessary. If the result is negative or indefinite, the final reading should not be made until the end of an hour's incubation. Bloods often will not show clear-cut reactions until fifty to sixty minutes incubation. At the end of one hour, the suspension is centrifuged at 500 R.P.M. for one minute. This packing sharply accentuates the agglutination. The mixture is resuspended by shaking, poured on a slide and examined immediately under the microscope. The long period of incubation often concentrates the serum so that the viscosity is increased and if the mixture on the open slide is left to stand for any length of time, pseudo-agglutination may be so marked that an interpretation is very difficult. Agglutination of the cell suspension indicates an Rh positive grouping. The cells from different individuals show a tremendous variability in the degree to which they clump. The same serum will give results varying from complete agglutination to slight clumping of two or three cells.

The cross-matching of the patient's serum with donor's cells is done in the same manner. The avidity of cells to agglutination varies greatly and often a weak serum will agglutinate only one

of many cell suspensions. In checking a patient's serum for antibodies it is necessary to cross-match against as many suspensions of group compatible cells as possible. If even one strain of cells is agglutinated the presence of Rh agglutinins is almost certain.

TABLE I. IRREGULAR AGGLUTINATION OBSERVED WITH DIFFERENT STRAINS OF Rh ANTISERA

	Serum			
Cells	L	M	C*	C2*
Mrs. C.	Plus	Plus	0	Plus
Mrs. T.	Plus	Plus	Plus	0

*C and C2: two separate strains of commercial antisera.

There is another property of the Rh antibody which is either identical with or related to the characteristic just described. A given strain of serum may yield a negative, or faintly positive, reaction with some cells and yet another serum, apparently of equal potency, may give a positive reaction. In other words, cells which are negative with one serum may be positive with a different strain of serum. This phenomenon has been observed by practically every investigator.[5,8,15,16,17] The commercial sera available to the average hospital laboratory also manifest this irregularity of agglutination (Table I).

The significance of this variability in clinical practice is very great. The cells of a prospective donor may be Rh negative with one strain of serum and yet actually be Rh positive. If such blood should be given to a sensitized patient, a reaction probably would occur. Before individuals are finally classified as Rh negative, the cells should be checked against several strains of serum. At least the two available commercial strains should be on hand in every laboratory.

Appearance of Antibodies.—The time when antibodies appear during pregnancy, or after the initial tranfusion of Rh positive blood, determines when reactions will occur. A study of pregnant women to establish a prenatal diagnosis of erythroblastosis or in preparation for transfusions has given some information relative to the time antibodies may be expected in pregnancy. This data is summarized in Table II.

It is apparent from Table II that antibodies may be present as early as the tenth week of pregnancy. If these sensitized women should be transfused with Rh positive blood, either because of anemia or a loss of blood attending an abortion, they would develop a reaction. The following two summaries illustrate this type of reaction.

TABLE II. APPEARANCE OF ANTIBODIES IN PREGNANCY

Patient	Weeks of Gestation*	Antibodies
W	12	Marked
C	8-10	Trace
La	12	Marked
B	(a) 6 (b) 10	0 Trace
M	(a) 10 (b) 14	0 Definite

*Historical.

Case 9.—This patient aborted at about twelve weeks of pregnancy. Because of anemia she was transfused from a sister. There was no reaction. About the tenth day after the abortion she was transfused from the husband. She developed chills and fever. Jaundice and oliguria were present on the next day. The oliguria improved and the patient finally recovered. The mother was a group A and Rh negative. The husband was a group A and Rh positive. The bloods were sent for study a few days after the transfusion. None of the donor's Rh positive cells remained in the patient's circulation. The serum strongly agglutinated all Rh positive cells.

Case 10.—This woman suffered an abortion at about ten weeks of pregnancy. She was admitted to the hospital for a transfusion. The admitting diagnosis was "secondary anemia" and no information was given to the laboratory relative to the recent pregnancy. No Rh studies or Levine cross-matching was done. Group compatible blood was given and the patient developed distress, fall in blood pressure and chills and the transfusion was discontinued. The blood studied a few days after the transfusion was Rh negative, all the donor's Rh positive cells were gone and Rh antibodies were present in low titre.

Comment.—These two cases are typical examples of reactions occurring from transfusions of Rh positive blood to women sensitized early in pregnancy. In Case 10 oliguria was present illustrating that such reactions may be severe.

The possibility of sensitization in very early pregnancy as in ectopic pregnancy has not yet been established. Hamilton and Martini[9] reported an increased hazard from such transfusions. In the course of this study there has been only one Rh negative woman with an ectopic pregnancy and this one was given three transfusions of Rh positive blood without reaction.

The time at which antibodies appear after transfusion was studied in seven cases and the results are summarized in Table III.

TABLE III. APPEARANCE OF ANTIBODIES AFTER TRANSFUSION

Patient	Positive Blood c.c.	Day of Appearance after first Transfusion
S	500	6
P	1,000	4*
F	2,500	28
C	2,200	33
G	1,500	14
A	1,750	32
H	1,500	9†

*Four months pregnant. Reaction developed on fourth day with the third transfusion.

†Ectopic pregnancy. Transfused three times in first two days without reaction.

In these few cases antibodies were demonstrable either by laboratory means or by transfusion reactions at times varying from four to thirty-three days. While this data is based on only a few cases, it nevertheless distinctly indicates that sensitization may develop as early as the fourth day after the initial transfusion. In one of Wiener's cases antibodies appeared on the fifth day.[23] In the case cited here, with antibodies on the fourth day, the patient was pregnant which may have hastened the appearance of the antibodies.

The persistence of antibodies after the sensitization stimulated by pregnancy has been studied quite extensively. Levine found that 50 per cent of the cases lost their antibodies within six months but that a few retained the sensitization for as long as 18 months.[16,18] Potter, Davidsohn, and Crunden[21] reported a case in which antibodies were demonstrable six years after the last pregnancy. In the group of patients in this study one had antibodies thirty months after the last pregnancy. Another is still strongly sensitized fifteen months after her pregnancy. In one case antibodies reappeared during a respiratory infection even though they had not been demonstrable during the prior six months. In one other case there was a distinct increase in antibodies associated with an intercurrent infection.

The titre immediately after the pregnancy does not determine the duration of demonstrable antibodies. One patient with a postpartum anti Rh titre of 1-5,000 showed no antibodies three months later, while another patient with a postpartum titre of 1-120 still is strongly sensitized at fifteen months.

Because of this persistence of Rh antibodies for long periods of time after a pregnancy, it seems advisable to inquire into the obstetrical history of every woman before a transfusion is given. The child born of a strongly sensitized woman should show some evidence of erythroblastosis: prematurity, stillbirth, neonatal death, edema, jaundice, hemorrhage or anemia. When the obstetrical history reveals any such abnormality the Rh status should be determined and studies made to determine whether or not antibodies are still present.

The Reaction

In the test tube the union of Rh antigen and antibody results in agglutination only and in some cases this agglutination is very slight. On the other hand, in case of erythroblastosis, the union of the maternal antibodies with the fetal antigen produces hemolysis, capillary injury and necrosis of cells in the liver and brain. The kidneys of the fetus are not significantly damaged. When antigen and antibody are combined in the circulating blood as occurs when Rh positive blood is transfused into a sensitized individual, there occurs a marked hemolysis of the transfused cells and often oliguria or anuria. The interference with kidney function may be very marked but the nature of the kidney lesion is not clearly understood. It has been customary to attribute the anuria to obstruction of the kidney tubules by hemoglobin. However, in many cases the hemoglobin deposits have been insignificant or absent. Tubular degeneration similar to that caused by bichloride of mercury poisoning has been observed. Necrosis of the entire cortex has been described.[9] Since cellular damage is such

a prominent feature of erythroblastosis, it seems very probable that in transfusion reactions of this type cellular injury to the tubular epithelium is a significant part of the kidney lesions and may be fully as important, if not more so, than tubular obstruction by hemoglobin.

In most of the cases reported in the literature the reactions have been severe or fatal, probably because minor reactions are not reported.[3,4,9,15,16,17,20,22,23,25] In the course of the studies reported here only two serious reactions were observed and both patients recovered. Many cases of serious reactions reported in the literature prior to the time Rh studies were available may have been due to Rh sensitization. Dieckman[7] reported eight severe reactions at the Chicago Lying-in Hospital. Four of them were due to group incompatible blood and all of these four patient survived. In the other four patients the reactions occurred with compatible blood and may have been due to Rh sensitization. All of these four patients died. De Gawin[6] reported 3,500 transfusions with thirteen severe reactions. Six of these occurred in association with pregnancy or after multiple transfusions. In the recent literature many cases have now been reported with complete Rh studies. Hamilton and Martini[20] point out that a fatal reaction to a transfusion may be obscured by, or confused with a deterioration of the patient's condition due to the primary disease. The following case suggests such an occurrence.

Case 11.—A woman near term was admitted for so-called "apoplexy" of the uterus. A cesarean section and hysterectomy was done. She was transfused from the husband. A few hours after the operation she developed jaundice and a high fever and she died about twelve hours later. The patient's condition throughout had been critical.

Comment.—No studies of the blood were made in this case. It is, therefore, impossible to determine whether or not the jaundice, fever and death were due to a transfusion reaction of the Rh type or to a natural deterioration in the patient's conditions.

Management of Transfusions During Pregnancy

Transfusions to women during their pregnancy or puerperium must be governed by the following considerations. The sensitization developed by some Rh negative women may appear early in the first trimester and may persist for many months after the delivery. It may be that during periods of fever and illness these antibodies become more active. The child born of such a sensitized mother usually shows some manifestation of erythroblastosis. However, if the woman is a primipara there is no prior history to suggest that she may be sensitized. Even if a woman has previously given birth to one or more normal children, she may be sensitized during the current pregnancy. In one case studied the woman had given birth to five normal children. The sixth pregnancy resulted in a stillborn, erythroblastotic baby. She was Rh negative and her serum contained anti-Rh agglutinins. Sometimes the erythroblastosis in the child may masquerade as a death due to prematurity, eclampsia, anoxemia or other causes even after an autopsy study. Often the erythroblastotic baby may appear quite normal at birth or for several days. It is, therefore, almost impossible for the physician to be completely certain that a woman is not Rh negative and sensitized.

Except in extreme emergencies no woman who is pregnant, or recently delivered, should be given whole blood until her Rh status has been determined. In almost all emergencies plasma or serum can be given until the Rh tests can be prepared. This applies even in cases of early abortions but probably not in the usual ectopic pregnancy.

If the woman is proven to be Rh positive, transfusions of any group compatible blood can be given and the question of Rh sensitization can be dismissed. If the woman is Rh negative and if the baby has died or is abnormal in any way, the possibility of erythroblastosis is very great and it is almost imperative that Rh negative donors be used. If, however, the mother is Rh negative and the child is not yet born, or is normal, a troublesome situation is present. Such a woman may not be sensitized.

The serum can be studied for the presence of Rh antibodies and if any are demonstrated only Rh negative donors can be used. If no agglutinins are demonstrable, there still exists the good possibility that this particular individual has weak or absent agglutinins but does possess hemolysins or cytotoxic antibodies which cannot be demonstrated. It is, therefore, safer to use Rh negative donors for all Rh negative women during pregnancy in all circumstances if such donors are available.

Sometimes no Rh negative donors can be found. In such cases the biologic test proposed by Wiener, Silverman and Aronson[27] may be carried out. This test consists of the intravenous injection of 50 c.c. of the prospective donor's blood with a determination of the icteric index before and one hour after the injection. If no hemolysis occurs as demonstrated by an elevation of the icterus, a second 50 c.c. should be given if time allows. The icterus should be determined one hour after this second injection. If no elevation of the icterus occurs, the remainder of the transfusion can be given. The following two summaries illustrate the use of this test.

Case 12.—The woman was admitted to the hospital during the course of an inevitable abortion at four months of pregnancy. She had had a secondary anemia prior to the pregnancy and had been given a transfusion about one year before. Her blood was group 0 and Rh negative. The prospective donor was the one who had given blood to the patient a year before. His blood was group 0 and Rh positive. There were no other donors and it was desired to transfuse the patient if possible. The Levine cross-matching showed no agglutination. A Wiener biologic test was carried out and was supplemented by hourly temperature readings. The results are summarized in Table IV.

TABLE IV. POSITIVE WIENER BIOLOGIC TEST

Hour	Blood c.c.	Icterus	Temperature
9:00		2	98.6
9:30	50	—	98.6
10:30		6	99.2
11:30	50	—	99.2
12:30		8	100.8
2:30		6	101.2
4:30		—	102.0

Comment.—In this case, even though the Levine cross-matching showed no incompatibility, 50 c.c. of the prospective donor's blood raised the icterus from 2 to 6; 100 c.c. raised the icterus to 8 and induced a fever of 102 degrees. Such a reaction from 100 c.c. of blood indicates that the usual 500 c.c. would have resulted in a severe reaction.

Case 13.—This woman was admitted to the hospital because of toxemia and anemia. A cesarean section was anticipated and the physician desired to give several transfusions. The patient was a group A and Rh negative. The Levine cross-matching with eight different group compatible Rh positive cell suspensions showed no agglutination—*i.e.*, there were no demonstrable antibodies. One Rh negative donor was found and all others were Rh positive. A Wiener biologic test was carried out and the results are given in Table V. Because the test was negative, 500 c.c. of the blood was given. A second transfusion of Rh positive blood was given a few days later. Ultimately a cesarean section was done and living twins were delivered. There was no evidence of erythroblastosis and the mother developed no demonstrable Rh agglutinins during her hospital stay.

TABLE V. NEGATIVE WIENER BIOLOGIC TEST

Time	Blood c.c.	Icterus	Temperature
10:00		2	98.6
10:30	50		98.6
11:30		2	98.6
12:00	50		98.6
1:00		2	98.6
3:00	400		98.6
4:00		2	102.0*

*Pyrogen reaction.

This test proposed by Wiener, Silverman and Aronson seems very useful and probably will allow the physician to transfuse some patients who otherwise would have to be treated by other less efficient means. The test is new, and more experience with its use is necessary.

The persistence of Rh antibodies for months or years after a pregnancy presents another problem. Such sensitized women would suffer a reaction if, because of anemia, shock or blood loss, a transfusion of Rh positive blood should be given. If there is any history suggesting erythroblastosis in the babies, the Rh status should be determined. If the woman is Rh negative, tests for antibodies or a Wiener biologic test should be done before the transfusion is given. Better still, Rh negative donors should be used if they are available.

Management of Multiple Transfusions

The planning of a program for a patient who is to receive several transfusions is greatly simplified if the Rh grouping is known. If the patient is Rh positive the entire question of sen-

sitization can be dismissed. If, however, the patient is Rh negative, sensitization may develop and consideration must be given to this possibility. It has been shown that there is a period after the first transfusion before antibodies appear. The duration of this latent period is not known for any one patient but it may be as short as five days.

In the majority of patients it is usually quite evident whether a single transfusion or several transfusions will be necessary. In many of the cases where multiple transfusions will be used it would be just as efficient to give all the blood within a period of five or six days. In other words, in many patients the entire transfusion program can be crowded into the first few days of hospitalization and the possibility of sensitization can be avoided.

Case 14.—This patient while in the hospital developed a massive hemorrhage from a duodenal ulcer. The hemoglobin dropped to 6.3 grams. The patient was a group A and was Rh negative. Several transfusions could be anticipated and it was the intention to do a gastric resection whenever the patient's condition permitted. Many donors were available but only two were Rh negative. During the next six days the patient was given 3,000 c.c. of blood and a gastric resection was done on the sixth day. There were no reactions and the patient's postoperative course was excellent. No more transfusions were necessary. 2,500 c.c. of the blood was Rh positive and 500 c.c. was Rh negative. One Rh negative donor was held in reserve to be used if necessary during the period when sensitization could be expected.

Comment.—In this case the need for many transfusions was obvious. Knowing that the patient was Rh negative and might develop reactions after the first five or six days, the transfusions were crowded into the first few days and the operation was done on the sixth day. An Rh negative donor was held in reserve to be used if necessary during the period of possible sensitization. This patient developed traces of antibodies on the twenty-eighth day after the first transfusion.

Case 15.—This patient had shock and anemia from a ruptured ectopic pregnancy. She was a group 0 and Rh negative. All available donors were Rh positive and all were compatible by the Levine cross-matching. Sensitization occurring during the course of the ectopic pregnancy was dismissed as improbable. On the day of surgery she was given one transfusion of 550 c.c. of Rh positive blood without reaction. On the following day her condition was satisfactory but since she had lost considerable blood, she was given two more transfusions of Rh positive blood—both without reaction.

Comment.—The transfusions on the day after operation were not imperative. It was felt that her convalescence would be hastened by replacing the blood lost. Because she was an Rh negative individual, all the desired transfusions were given promptly before sensitization could develop. On the ninth day this patient developed antibodies against Rh positive cells.

In some cases it is neither desirable or permissible to crowd the transfusions into a short period of time. In others the blood loss is continuous and transfusions over a long period are necessary (Case 8). Most blood dyscrasias require transfusions over a long period.

Case 16.—This woman was admitted to the hospital because of fever and weakness. A diagnosis of ulcerative endocarditis due to S. viridans was established. This patient was a group B and Rh negative. Multiple small transfusions were given. All the donors were Rh positive. The results of these transfusions are summarized in Table VI.

TABLE VI

Date	Blood c.c.	Days Since First Transfusion	Days Since Last Transfusion	Reaction
1/11/43	200	0	0	0
1/11/43	250	0	0	0
1/20/43	300	9	9	0
1/25/43	300	14	5	0
2/ 2/43	500	22	8	0
2/11/43	200	31	9	0
2/12/43	200	32	1	Chill
2/15/43	300	35	3	Severe chill

Rh antibodies demonstrable four days after last transfusion.

In Case 16 the transfusions were given at relatively short intervals. The reactions, when they developed, were of moderate degree. The second was more severe than the first. Experience with other similar situations suggests that if the transfusions are spaced close together the abrupt occurrence of a severe reaction may be prevented.

When antigen is introduced into a sensitized individual, it unites with the antibody and for

some time thereafter no antibody is demonstrable.[2] This absence of antibodies immediately after a transfusion of Rh positive blood has been called a "negative phase" by Wiener.[23,27] The antibodies reappear within three to five days. Theoretically it should be possible to completely prevent reactions in Rh sensitized individuals by giving transfusions at two- to four-day intervals and the use of closely spaced transfusions has been suggested as a means of preventing reactions.[23,28,29] That this is not true is illustrated in Case 16 and also Case 8. However, the reactions which developed in these two cases were not severe.

When transfusions to Rh negative patients must be given over a long period of time, it appears that the transfusions should be spaced at relatively short intervals. It appears probable that if the transfusions are given close together, the reactions, when they develop, will be mild and will serve as a warning to discontinue the use of Rh positive blood. Cases reported in the literature and Case 1 cited here indicate that if there are intervals of weeks or months between transfusions, the first reaction may be severe.

There sometimes occur situations wherein it is not possible to foresee the need for further transfusions. A patient may be given one transfusion and after an interval of several days the condition is such that another transfusion is desirable. In this case the laboratory workers are placed in a very difficult situation. It is often impossible to determine the patient's Rh grouping at this time. The blood cells from a compatible donor persist in the recipient's circulation for three to four months.[15] If an Rh negative patient is given Rh positive blood, these positive cells persist in that patient's circulation. If this mixture of bloods is tested with the Rh antiserum, the donor's positive cells will be agglutinated and the technician may erroneously conclude that the patient is Rh positive. Wiener's differential blood grouping[15] does not help in these cases. It is impossible to distinguish between a weakly reacting Rh positive individual and an Rh negative patient who has received Rh positive blood. Physicians are urged to request an Rh grouping before the first transfusion whenever there is any possibility of more transfusions later. Technicians can also make it a practice to preserve, in the refrigerator, the original blood sample as clotted blood so that they may go back to it to determine the Rh grouping at a later time.

If in these cases the patient's Rh grouping cannot be established the physician may give a small trial transfusion or the Wiener biologic test transfusion can be done.

Transfusions to Erythroblastotic Babies

Transfusions to infants differ in no essential manner from those to adults. In the case of the baby with erythroblastosis, certain features complicate the situation. These babies have been exposed to maternal Rh antibodies which have become fixed to the child's tissues. Transfusion of the mother's blood adds more antibody to the infant and aggravates the child's condition. In two cases referred for study the mother's blood was used and in both instances the child died.

Levine stated that Rh negative blood should be used for transfusing erythroblastotic babies. This is impractical in many cases. The delay necessitated by a search for negative donors will, in many cases, result in the death of the infant. Experience has shown that the use of any group compatible blood other than the mother's is suitable. Theoretically, Rh positive blood will help in destroying the maternal antibody fixed to the infant's tissues. The entire question of which Rh type of blood to use for erythroblastotic babies needs more study. However, in the meantime, the condition of the child should never be endangered by delays attending a search for Rh negative donors. The majority of erythroblastotic babies have been treated with Rh positive blood with excellent results.

In many cases a diagnosis of erythroblastosis is possible before the child is born and it is advantageous to have blood available for transfusion immediately after birth. In such cases blood can be drawn from a group 0 donor beforehand and stored in the refrigerator—to be kept ready for use. The blood group of the offspring cannot be forecast unless both parents are group 0. Even when both parents are group A the offspring may be group 0 and the blood of the father may be incompatible. Therefore, only group 0 blood can be used without cross-matching.

Summary

The Rh substance is, in general, similar to the A and B substances which determine the division into the four blood groups. Rh positive indi-

viduals cannot become sensitized. Rh negative individuals, if inoculated by transfusion—or during pregnancy from the fetus—may develop antibodies. The Rh substance varies greatly in its antigenic property: some people become sensitized very readily, while others either form no antibodies or do so only to a slight degree. The antibody which can be demonstrated in the laboratory is the agglutinin. Apparently other types of antibodies are formed which are not detected by laboratory tests but will produce transfusion reactions or erythroblastosis in the fetus. The absence of demonstrable agglutinins, therefore, does not exclude the presence of other dangerous antibodies.

In pregnancy the antibodies may develop between the eighth and twelfth weeks so that transfusions after early abortions must be handled in the same way as those later in pregnancy. The antibodies may persist for many months after delivery and in exceptional cases have been observed at two and six years after the last pregnancy. An investigation of the obstetrical history of women should be made prior to a transfusion and, if any evidence of erythroblastosis in the babies is present, the possibility of sensitization should not be overlooked.

Except under rare circumstances whole blood should never be given to pregnant or puerpural women until their Rh grouping is known. If they are Rh negative, only Rh negative donors should be used. In case where no evidence of sensitization can be demonstrated, Rh positive blood can be given if the Wiener biologic test is negative.

If multiple transfusions are to be given a knowledge of the Rh grouping is very helpful. If Rh positive, the transfusion program can be carried out with no worry relative to Rh sensitization. If the patient happens to be Rh negative, an attempt may be made to complete the transfusion program within the five- or six-day period before antibodies can develop. If this is not practical, the transfusions should be spaced close together—for example, at two- to four-day intervals. The evidence indicates that if transfusions are spaced close together, the reaction—when it develops—will be of a mild degree. After reactions develop, the transfusion program can be continued but only by using Rh negative donors.

In transfusing erythroblastotic babies the mother's blood should not be used. There may be a question as to whether or not Rh negative donors are preferable but under no circumstances should the child's condition be allowed to deteriorate because of a delay attending a search for Rh negative donors.

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CLINICAL-PATHOLOGICAL CONFERENCE

MINNEAPOLIS GENERAL HOSPITAL

A. J. Hertzog, M.D., and S. V. Lofsness, M.D.
Pathologists

Presentation of a Case
A-43-1788

DR. PETIT: The case is that of a Mexican, fifty-two years old, who was admitted to the Medical service on August 28, 1943, where he died on September 23, 1943.

On admission he complained of a cough of three to four years' duration. This was usually worse in the winter, but was present the year around. He raised small amounts of mucus, which was occasionally bloodstreaked; he never coughed up any appreciable amount of blood. During the last three months, the cough became more severe; he had attacks of severe coughing many times each day, each of which lasted several minutes, during which he would raise moderate amounts of mucus. He had also noted increasing difficulty in swallowing these last three months; food often seemed to stop in his chest and caused some discomfort. He gradually ate less food and lost 20 pounds in weight during this period. He became short of breath on slight exertion. There was a sensation of numbness in his left arm and hand during some of the coughing spells.

Examination showed a middle-aged Mexican, ambulant, and not acutely ill. His temperature was 98 degrees, pulse rate 70, respiratory rate 20, blood pressure 100/70. The only abnormal findings were in the chest; there was dullness to percussion behind the upper part of the sternum and extending laterally for about 5 cm. between the first and third ribs.

Laboratory studies: Hemoglobin 95 per cent; RBC, 5,120,000; WBC, 5,400 with 82 per cent neutrophiles, 15 per cent lymphocytes and 3 per cent monocytes; urinalysis, negative; Rytz, Kline and Wassermann were positive.

DR. STENSTROM: The x-rays of his chest taken on admission show a dense mass about 12 cm. in diameter in the superior mediastinum. It is bilateral, but more prominent on the right. A lateral view shows it is anterior, and in contact with the upper portion of the sternum. It could be either a sub-sternal goitre, an aneurism, or a neoplasm.

DR. PETIT: The patient remained ambulant and afebrile for three weeks. He continued to have coughing spells frequently; his cough was harsh and brassy, and usually he raised a little mucoid sputum. The Rytz, Kline and Wassermann tests were repeated a few days after admission and were again positive. Ten sputum samples were examined for tubercle bacilli and all were negative. About two weeks after admission he was given a moderate dose of deep x-ray over the tumor. This was done as a diagnostic procedure, feeling that the tumor would rapidly decrease in size if it was a lymphoblastoma. However, the tumor remained the same size.

DR. STENSTROM: Further x-ray studies of the tumor were done. The mass displaces the barium-filled esophagus posteriorly very much, and this explains his dysphagia. An over-exposed film shows no evidence of teeth or bones in the tumor, as sometimes seen in teratomas. The arch of the aorta seems to be distinct from the mass. The mass does not pulsate. We were not able to determine whether this mass was a neoplasm or an aneurism.

DR. PETIT: Because the tumor seemed to be distinct from the arch of the aorta, very high in the chest, and non-pulsatile, most of the men who saw this patient felt that it was a neoplasm, and exploratory operation was being considered. However, Dr. Moses Barron thought that it was very probably a round, thrombosed aneurism. On September 21, 1943, his temperature rose to 100.4 and his coughing increased, with periods of dyspnea and cyanosis. He was given morphine and oxygen, and tracheal suction was used. His temperature rose, the dyspnea and cyanosis increased, and he died on September 23, 1943.

Autopsy Findings

DR. PETIT: The body was slim but muscular. The only important findings were in the chest. The heart weighed 330 grams and appeared normal. The mass in the mediastinum was an aneurism, which arose from the superior surface of the arch of the aorta just proximal to the innominate artery. The aneurism was round, 12 cm. in diameter, and communicated with the aorta by an opening only 2x1 cm. The aneurism had a thin fibrous wall, but was completely filled by a laminated thrombus. The root and arch of the aorta showed a white, wrinkled intima, very suggestive of syphilitic aoritis. The lungs were heavy, the right weighing 670 grams and the left 1,000 grams. Both showed marked edema and congestion of the lower and posterior portions, with small patches of consolidation.

DR. LOFSNESS: Microscopically, the arch of the aorta shows collections of lymphocytes in the adventitia and

(Continued on Page 1003)

HISTORY OF MEDICINE IN DODGE COUNTY

BY JAMES ECKMAN
Rochester, Minnesota

and

CHARLES E. BIGELOW, M.D.
Dodge Center, Minnesota

(Continued from October issue)

Dr. James (or Jacob) **Manning Saunders** (1816-1904) was born in Brookfield, New York, on April 11, 1816, the son of August Saunders and Eunice Lewis Saunders.[53] Not much is known about his early years, but it is believed that as a young man he supported himself by teaching school in New York State. He also, at some time, learned the trade of carpentry. On April 3, 1845, he was married to Miss Ellen A. Babcock (1825-1885), a native of New York State. She was a practical nurse of some experience, and later aided her husband considerably in his professional duties in Dodge County. She died of pulmonary tuberculosis in Dodge Center on April 29, 1885.

Dr. and Mrs. Saunders moved to Hamilton, New York, after their marriage in 1845, and in about 1850 or 1851 went to Milton, Wisconsin, which was a center for Seventh-Day Adventists, of which both the physician and his wife were members. There he worked at his trade of carpentry, and he may also have practiced medicine to some extent. In October of 1870 Dr. and Mrs. Saunders came to Dodge Center, which at the time contained and still contains a considerable number of Seventh-Day Adventists. They remained in Dodge Center for the rest of their lives. In 1871, at the age of fifty-five years, Doctor Saunders received the degree of Doctor of Medicine from the Hahnemann Medical College and Hospital of Chicago.[53] In the same year he became a charter member of the old Southern Minnesota Homeopathic Medical Society, and he was also elected to membership in the Minnesota State Homeopathic Institute.[53] He was elected second vice president of the institute in 1877, and first vice president in 1879.[53] His professional card, as printed in the *Dodge County Republican* of October 10, 1873, was as follows:

J. M. Saunders, M. D.
Homeopathic Physician
Pulls teeth without pain. Fills family cases.
Specialist in obstetrics and children's diseases.

Doctor Saunders was licensed to practice medicine in Minnesota on December 28, 1883.[53] By that year he had more than fulfilled the requirements for licensure by exemption of the medical practice act of 1883.[95] He registered his certificate in Dodge County (as required by law) under the name of "Jacob M. Saunders," but several persons who knew him have said that he never was known by any name other than "James M. Saunders." Possibly he anglicized the German "Jacob" to "James" for professional reasons. It has been said

that he was never referred to by anyone, including his wife, by any name other than "Doctor Saunders."

Ellis,[240] who knew Doctor Saunders personally, reported that before his wife died in 1885, the physician mingled freely with the people of Dodge Center; but that in later years he became almost a recluse, seeing no one but his patients and rarely venturing from his home in Dodge Center. Most of those who remember him have said that he was a dentist, rather than a physician, but without doubt he practiced medicine frequently. Doctor Saunders' health deteriorated rapidly after 1900, and he died on the farm of his daughter in Ashland Township in Dodge County on January 14, 1904, after a paralytic stroke.[241] He was buried at Milton, Wisconsin.[53]

Dr. Christopher Porter Gibson (1848-1933) was born in Ayer Township, Dominion of Canada, on October 26, 1848, the son of William John Gibson and Martha Stockman Gibson. Both his parents were Scotch-Irish, and both had been born in Ireland.[242] When he was about six years old, Doctor Gibson was brought to Dodge County by his parents, who had acquired a farm in the vicinity of Claremont. Doctor Gibson's daughter[242] said that as a small boy her father had run away from home to try to enlist in the Union forces during the War of the Rebellion, but that he was too small and too young to be accepted. He did, however, ride about Dodge County on horseback, carrying messages from the front to various residents in the county as they were received in the telegraph office. The boy was educated partly at the old seminary at Wasioja, and partly at the Owatonna High School. On November 15, 1871, while he was studying medicine on his father's farm, he was married to Miss Ione Ingalls, of Dodge Center. He and his wife returned to the farm, where he did farm work and studied medicine. On September 24, 1872, Dr. and Mrs. Gibson went to Chicago, where the young man enrolled at the Chicago Medical College, from which he was graduated in 1873. He then settled in Dodge Center, the home of his father-in-law, where, as previously mentioned, he became the partner of Dr. James A. Garver (1814-1901). In 1882 he helped draft the constitution and fee bill of the Dodge County Medical Society,[184] and in the same year, his daughter[242] reported, Doctor Gibson moved to Fairpoint in Goodhue County, but by the fall of that year he was in Concord. He was licensed to practice medicine in Minnesota on April 19, 1884, receiving License No. 878.[40] Doctor Gibson bought a house in Concord in 1884,[243] became superintendent of the Sunday school and deacon of the Christian Church in that town, and began to develop his interest in ornithology and taxidermy, which were his avocations throughout his life.

In September of 1888, it was noted,[244] Doctor Gibson made a trip to Colorado, but he was back in Concord before the end of the month.[245] It was believed that he could not endure the rarefied atmosphere of Colorado, and he never applied for licensure in that state.[246] It was evident, at any rate, that he had determined to leave Concord, for he soon sold his house in that village and moved to Dodge Center.[242] On April 4, 1889, he settled in Redwood Falls in Redwood County, where he was to practice medicine for twenty-three years. In Redwood Falls his practice extended for a radius of thirty miles of the town, and he was a highly successful physician. The demands on his strength, however, were so severe that his heart was affected by the strain. In 1912 his health had deteriorated sufficiently for him to seek rest, and after repeated serious episodes of cardiac distress he retired from the practice of medicine and bought a 380-acre farm some six miles north of Milaca in Mille Lacs County, to which he

removed.[242] There he began the breeding of fine livestock and also indulged his fondness for growing flowers of all kinds. When the United States entered the first World War, however, Doctor Gibson interrupted his tranquil retirement by joining the Medical Reserve Corps of the United States Army at the age of sixty-nine years. He became first a lieutenant and then a captain, and served at Fort Benjamin Harrison. In about 1923 Dr. and Mrs. Gibson moved to California, but in 1926 they returned to Minnesota to live in Minneapolis, where they occupied a residence at 2002 Fifth Avenue South.[40] After they had become more and more enfeebled, they decided to register at the Masonic home at Bloomington, Minnesota, which they did in 1932. Doctor Gibson died there of pneumonia and arteriosclerosis on March 20, 1933, at the age of eighty-four years.[247,248] At his death it was said[248] that he was one of the oldest members of the American Legion in the United States. His daughter[242] reported that so far as she could remember, her father never lost his temper on any occasion, and he was known to be one of the most highly regarded citizens in Redwood Falls and the county. His lifetime collection of stuffed and mounted birds, said to be one of the finest in the state, now reposes in the Redwood Falls High School.[242]

Dr. Horace P. Porter (1838-1912) of Claremont was one of the most restlessly energetic physicians who ever settled in Dodge County. Records of the Bureau of Vital Statistics of the Missouri State Board of Health,[249] in which state Doctor Porter died, indicate that he was born at Ellington, Connecticut, for which a township in Dodge County was named,[17] on February 6, 1838, the son of Philo Porter and Clarissa Skinner Porter. On his father's side Doctor Porter's ancestry extended back to the Seventeenth Century in America. Details of his early years are not known, but he was graduated from the Medical Institution of Yale College in 1861, and on September 17, 1861, became assistant surgeon of the Seventh Connecticut Regiment of Volunteers, a post which he occupied until March 19, 1864.[204] At the latter date he resigned so that he could accept the commission of surgeon to the Tenth Connecticut Regiment of Volunteers, which he did on May 1, 1864. He remained with this regiment until November 5, 1864. From November 14 to December 14, 1864, and from October 20, 1866, to August 22, 1867, he was an acting assistant surgeon under contract in the Regular Army of the United States.[204] In 1868 Doctor Porter was in Kalamazoo, Michigan, where on February 11, 1868, he helped to found the old Kalamazoo Medical Association.[250] In 1873, as previously recorded,[55] he appeared in Kasson, Minnesota, to which his younger brother, William E. Porter, had gone in 1865. There Doctor Porter investigated the possibilities of locating in some town along the road of the Winona and Saint Peter Railway Company, but he remained in Kasson for about one year. In September of 1874 he moved to Claremont,[56] where in addition to engaging in the practice of medicine, he sold blank deeds, mortgages and leases, and acted as the subscription agent for the *New York Tribune*.[59] In December of 1874 he began to publish one of the most curious frontier newspapers in the history of the state. This was *The Wind Mill* of Claremont, the first newspaper that village ever had. Excerpts from this odd four-page sheet have been printed herein previously, but a few more will be reproduced to show the ready wit and inventiveness of their author. The little publication had been in existence for about a year when Doctor Porter decided to suspend it for a while. He wrote:[59]

GOOD NIGHT, FRIENDS

This issue of our paper will be the last we shall publish this season. We announced that we should issue when "we feel like it," consequently we are under no obligations to appear regularly, as is the case with the older and more fully developed papers. We are going to hibernate during the winter that is now upon us; in short, we shall sleep the sleep of a just newspaper made perfect. In the spring, when the snows are melted from the ground, and the green grass appears, and the birds come back from the sunny south, we shall again come before our friends fresh from a restful and unconscious slumber, prepared to grapple and struggle mightily with a discriminating environment of numberless readers. Remember, friends, that THE WIND MILL is not dead, but is merely reposing in the arms of *"somnus,"* renewing its strength for the future time of need, the good time coming, when you will conclude to liberally sustain a weekly paper printed in Claremont.

In an "extra" edition of *The Wind Mill,*[251] issued in May of 1876, he exhorted his fellow townspeople of Claremont thus:

SOLEMNITIES

It will pay in the long run to patronize your own town. The west "slice" of Dodge county belongs to Claremont, and we *will* have it, with a big "chunk" from the eastern part of Steele. Geography don't lie, and the people are beginning to see it. Two years ago the jackals howled in the streets, and now we are securing a lively trade. . . . Now in Claremont the epidermis of things is a little tough, but the business interior, the vital organs of trade, are in a healthy condition. If you doubt it, bring in your country produce and give us a trial.

"You can buy anything from a first-class ocean steamship to a sperm whale in Claremont," he boasted in the same issue.[251] In another edition of *The Wind Mill*[60] he announced that: "Doctor Porter extracts teeth. Call and see him, and get rid of that old 'grinder' that has been keeping you awake for a week," and he took notice of his past business as well: "Please call and pay Doctor Porter that little bill which is now overdue."[60] He observed that "THE WIND MILL is the best printed paper in Dodge County. The typographical excellence of this paper has been the admiration of good judges of such matters,"[60] and said, possibly with his tongue in his cheek, that "The WIND MILL never resorts to vile personal abuse. The editor prefers to save up his venom for a special tract, which he will issue at some future time."[60]

In December of 1880 Doctor Porter inaugurated another publication, called *The Cosmopolitan,* but this organ did not long endure.

In March of 1882 it was announced[113] that Doctor Porter was selling his chattels at auction. Soon thereafter he went to Corning, Missouri; but in November of 1882 he was back in Dodge County, visiting his brother, William E. Porter, of Kasson. It was suggested[114] at the time that Doctor Porter might remain in the county, but he did not do so. In February of 1883 he removed from Corning, Missouri, to Shubert, Nebraska, in Richardson County.[115] It was said[252] that he was licensed to practice medicine in this county. But Doctor Porter did not remain long at Shubert; in September of 1884 he returned to Kasson to visit his brother.[121] In October he decided to relocate in Claremont,[122] where he became manager of the Cleveland House, laid in a small supply of drugs which he kept for sale, and issued another publication called *The Q,* which appears to have been decidedly ephemeral. He was hardly well settled in Claremont when he determined to leave Dodge County once again. In April of 1886, as previously set forth, he traded his stock of drugs to Dr. Charles Willmar Coleman (1855-1925) for Kansas land,[124] and in May left for Kansas.[125] He was not, however, licensed to practice medicine in Kansas, so far as avail-

able records indicate.[126] In October of 1886 it was said that Doctor Porter was living in Du Bois, Nebraska,[134] and on February 4, 1889, he was licensed to practice medicine in Missouri,[76] receiving License No. 4050. He was licensed to practice medicine in Jefferson County, Texas, on January 30, 1903, and to practice medicine in the state of Texas on February 18, 1908.[253] In the latter year he was living in Port Arthur, Texas. He died at Butler, Missouri, on December 23, 1912, and at his death it was said that at one time he had served as surgeon general of the Grand Army of the Republic.[254]

Dr. Nils Schultz Holterman (1845-1895) was born in Trondhjem, Norway, on May 29, 1845, the son of Michael Holterman and Nicoline Holterman. The father was pastor of the second largest Lutheran church in the city of Trondhjem. The son, Nils, was one of five boys born to his parents: one of the sons (Christian) became a major general in the army of Norway; one (Lisco) became a sea captain; two (Marcus Frederick and Peter) died at the age of eighteen; and Nils became a physician.

The young man was educated in the public schools in Norway, and was subsequently graduated from what is now the Kongslige Fredriks Universitet Medisinske Fakultet at Oslo. During vacation periods he served as a medical attendant at a health resort in Germany, and in the course of his education he learned to speak German, English, French, Greek and Hebrew, in addition to Norwegian.

On July 28, 1868, Doctor Holterman was married to Miss Marie Krog of Copenhagen. Soon thereafter he immigrated to the United States with his wife. He planned to locate in Chicago, and in fact arrived in that city just before the great conflagration of 1871. Most of his possessions were lost in the disaster, and he himself served as a picket to protect the property of others from looters.

Next, Doctor Holterman moved to Saint Louis, where he attended the old Missouri Medical College, which in 1899 was absorbed by the present Washington University School of Medicine. In about 1874 he moved to Kasson in Dodge County, where he practiced medicine for four years.

In 1878 Doctor Holterman removed to Glenwood in Pope County. This was his home until his death, but for some years he had an office in Starbuck, at the western end of Lake Minnewaska, and he also traveled as far as Donnelly in Stevens County, on professional calls. He was licensed to practice medicine in Minnesota on December 31, 1883, receiving License No. 596.[40] From 1881 to 1883 he was coroner of Pope County. In June of 1892 he relinquished his office in Starbuck to Dr. Ole E. Linjer (1869-1907), who served as city physician of Minneapolis in 1906 and 1907.

Doctor Holterman died in Alexandria, Minnesota, on July 17, 1895. He was remembered as an intrepid physician who would travel as far as forty miles by skis to reach his patients, and as a self-effacing professional man who often would make sure that his patients received every kind of drug or medicine necessary, even though he might know that he would never be paid for either his services or his drugs.

(To be continued in the December issue)

References will appear at the conclusion of this article.

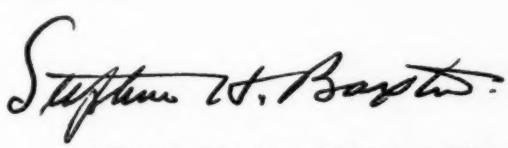
President's Letter

LOOKING INTO THE FUTURE

"The mind is so intimately dependent upon the condition and relations of the organs of the body, that if any means can ever be found to render men wiser or more ingenious than hitherto, I believe that it is in Medicine that they must be sought for. It is true that the science of Medicine, as it now exists, contains few things whose utility is very remarkable; but without any wish to depreciate it, I am confident that there is no one, even among those whose profession it is, who does not admit that all at present known in it is almost nothing in comparison with what remains to be discovered; and that we could free ourselves from an infinity of maladies of body as well as mind, and perhaps, also, even from the debility of age, if we had sufficiently ample knowledge of their causes, and of all the remedies provided for us by Nature."

Thus wrote Descartes in 1637. The statement that "Medicine, as it now exists, contains few things whose utility is very remarkable" could, without arrogance or undue self-conceit, be disputed today, but, with that exception, the quotation is as pertinent as the day it was written. If we are able at all to profit by the lessons of history and past experience, we must necessarily hold that opinion. Imagine the embarrassment, if he were living now, of the Commissioner of Patents who wrote, two hundred years after Descartes, and a hundred years ago, "We see the arrival of that period when human improvement is at an end"! The past fifty years have seen greater advances in medical sciences than were experienced in all the preceding history of the human race, but to think that the limit had been reached would show an entire lack of imagination. Cancer is no more mysterious to us than was tuberculosis before the time of Koch. Diphtheria was no more baffling before the discovery of Loeffler, than is rheumatic fever to us today. Who would have the temerity to say that the secrets of cancer and rheumatic fever will never be revealed? Within the memory of many of us, the practice of medicine and surgery has been revolutionized and, doubtless, researches in the field of chemistry and physiology will bring about equally radical changes in the future.

When men can learn the secret of living together in peace and can utilize the discoveries of science to promote human welfare and happiness, then medical science, also, will be able to concentrate all its talent and resources to the pursuit of the knowledge necessary for materializing the prophetic vision of Descartes. In the meantime, in spite of the interruptions and dislocations caused by war, medical men, whether on the battlefronts or in the laboratories, pursue their work of repair and restoration and of saving of life. Medical sciences alone, of all the sciences, are not diverted to purposes of destruction of life and property, but are devoted solely to the discovery and improvement of the means of combating disease, of saving life and of repairing the injuries caused by war.



President, Minnesota State Medical Association

Editorial

CARL B. DRAKE, M.D., *Editor;* GEORGE EARL, M.D., HENRY L. ULRICH, M.D., *Associate Editors*

WILL CONGRESS SWELL THE RANKS OF BUREAUCRACY?

THE Wagner-Murray Bill (S.1161) was introduced in the Senate on June 3, 1943. As every physician knows, this bill provides for a six per cent tax on employes earning up to $3,000 a year and a six per cent tax on employers for every employe earning up to $3,000 a year, a seven per cent tax on self-employed individuals, and a 3.5 per cent tax for all government employes. Of the twelve billion dollars so raised, a minimum of three billion will be used for the medical care and hospitalization of some 110,000,000 of the people in the country under the direction of the Surgeon General of the Public Health Service.

The people through their representatives in Congress will be asked to choose whether to take the practice of medicine out of the hands of the profession and place it in the hands of a bureau in Washington or not. If such a change will result in better medical care of the people of this country, will result in a lower mortality, will reduce the cost of medical care, and will stimulate initiative on the part of practitioners, the bill should be passed. We believe that its passage will do no one of these things.

Statistics of the U.S.P.H. Service show that in 1942, the death rate in this country was the lowest on record (10.3 per thousand). Figures given out by the Metropolitan Life Insurance Company on its experience with its millions of subscribers which always closely parallel those of the country at large, show an astounding reduction during the past twenty-five years from typhoid, diphtheria, scarlet fever, influenza, pneumonia, and tuberculosis, and a marked reduction even from cardiovascular disease, appendicitis, and puerperal conditions. Does this indicate the need for a radical change in the method of dispensing medical care in this country?

Bureaucracy is a very grave menace in our country today. For instance in Ohio, there are 90,000 federal employes to 25,000 state; Massachusetts 129,000 federal to 21,000 state; Pennsylvania 215,000 federal to 44,500 state; Wyoming 6,200 federal to 1,100 state. Federal employes at present outnumber all state, county, and municipal employes in the country, and 55 per cent of these federal employes are not directly employed in the war effort. If the Wagner-Murray Bill is passed, the cost of a greatly expanded bureau will be added to the actual cost of the medical care dispensed. And each additional employe of the U.S.P.H. Service will be added to the present 3,000,000 civilian employes of the federal government (exclusive of employes of the Army and Navy) who will receive medical care at half rate and whose livelihood will depend on continuation of the bureau. The passage of this bill will add great numbers to the already large number of federal employes, and they in conjunction with the almost equal number of state, county, and municipal employes will constitute a political block which, receiving special consideration in respect to medical service, will exert a tremendous power. We seem to be approaching a time when one-half of the people will be paying the other half to simply exert the functions of expanded government.

In an editorial on the Wagner-Murray Bill which appeared in August was appended a list of our Senators and Congressmen. They should now be addressed at Washington. Do not procrastinate.

SULFAMERAZINE

THE frequent occurrence of crystalluria and hematuria in patients treated with sulfadiazine has stimulated the search for a chemotherapeutic agent which might lead to fewer renal complications. Sulfamerazine (2-sulfanilamido-4-methylpyrimidine) has recently been investigated because of its greater solubility in water and urine.[7]

Several reports have been made concerning its use in a wide variety of human infections.[1,2,4,5,6] Sulfamerazine is more rapidly absorbed from the gastro-intestinal tract and is more slowly excreted in the urine than sulfadiazine. Therefore effective blood levels can be maintained with

smaller doses given at longer intervals than with sulfadiazine. Apparently, an initial doze of 4 Gm. followed by 1 Gm. every eight hours is sufficient for all but the most severe infections in adults. Sulfamerazine appears to be fully as effective against infections due to pneumococci, meningococci, streptococci and B. coli. Sulfathiazole continues to be the most effective sulfonamide in staphylococcic infections.

The incidence of crystalluria, hematuria and blockage of the renal tubules, pelves and ureters due to sulfonamide crystals does not appear to be any less with sulfamerazine than with sulfadiazine. The most effective means of prevention of renal complications during sulfonamide therapy continues to be the maintenance of a urine output of at least 1,200 c.c. per day. Considerable evidence has accumulated that the maintenance of an alkaline urine reduces the incidence of crystalluria during therapy with sulfamerazine and sulfadiazine. From 15 to 20 Gm. of sodium bicarbonate per day will keep the urine continuously alkaline.[3]

WENDELL H. HALL, M.D.

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BARBITURATE PRESCRIPTIONS

THE much needed federal law for the control of the dispensing of barbiturates is specific in its stipulations. Only physicians and dentists may prescribe for human consumption barbitol or any of its derivatives or preparations containing these drugs. In the case of refills of such prescriptions, written or verbal authority may be given the pharmacist, and in case of an emergency, an original prescription may be telephoned the pharmacist but must be followed by a prescription.

Pharmacists, as a rule, are willing to coöperate with the medical profession in filling these barbiturate prescriptions. Apparently they have been too willing, and the medical profession has not been coöperative in the sending in of written prescriptions when the barbiturates have been dispensed in presumable emergencies. The druggist is required to keep a separate file of these prescriptions and to make notations on them in ink when they are refilled on verbal order from the physician. These files are inspected periodically by federal agents, who are at present insisting that the laxity cease. The penalty of $1,000 fine or a year in jail or both is not pleasant for the pharmacist to contemplate as a penalty for accommodating his clients and the medical profession.

The barbiturate law is a good one and was passed in order to place the dispensing of these drugs in the control of the medical profession. The least we, as physicians, can do is to cooperate with the pharmacists in carrying out the provisions of the law.

ORDERS FOR INDUSTRIAL NURSES

INASMUCH as numerous requests have been received by the Council on Industrial Health of the AMA for reprints of its report entitled, "Standing Orders for Nurses in Industry," we are publishing the report in this issue under the section on Industrial Health as an aid to physicians in industry and to committees on industrial health in state and county medical societies.

The report is an outline which may be modified by industrial surgeons to meet their requirements.

The industrial nurse is a valuable assistant to the industrial surgeon. While her part in the medical care of employes should in general be restricted to the field in which she is trained, the surgeon can delegate certain duties to an intelligent trained nurse. Especially should she be instructed how to proceed in the carrying out of first aid treatment not only for minor but for major injuries. Most important is the nurses's ability to differentiate between situations which require the physician's attention and those which do not.

It is interesting to note that the Council does not approve the dispensing of vaccines and vitamins in the hope of preventing colds nor the dispensing of cathartics from an industrial medical department.

MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

BIG BUSINESS POINTS THE WAY

Everyone appreciates the appalling job ahead of industrialists and private employers, big and small, when the wheels of war industries come to a standstill and the men and women are mustered out of the services.

Everyone does not know, perhaps, that business leaders regard the job in the light of a last reprieve, the dragon which they must subdue, like the knights of old, or suffer annihiliation.

Gone is the easy assurance that failure at this job signalizes no more than a cause for regret and not the disappearance of an order and a way of life.

America at the Crossroads

"Looking backward to these times," says James H. McGraw, Jr., publsher of *Business Week*, "future historians are likely to say that here we Americans stood at the crossroads and, consciously or not, made our choice between a system of private enterprise and personal freedom and a system of collectivism and regimentation."

It is significant that business at the crossroads no longer relies upon mere opposition to stem the tide.

The fact is that the first steps toward government regimentation have long since been taken. Since 1932, America has become well accustomed to the idea that government can and will make jobs. After this war the government can and will make jobs again, if necessary, more of them at more levels. In so doing it will seal the fate of private business and individual enterprise. That is, unless private business is capable of providing the jobs. It is that possibility which has set the Committee for Economic Development upon a program of widespread activity.

If businessmen, by planning ahead and working together can get production going full speed ahead without much unemployment or for long—then government will have no excuse for providing the jobs or taking over the industries.

Committee at Work

The situation is seen clearly by astute leaders of big business, Paul G. Hoffman, president of the Studebaker Corporation, for one. Mr. Hoffman is chairman of the Committee for Economic Development which is now at work on the problem.

"All of us agree," Mr. Hoffman said in a recent address, "that the peace will most certainly be lost if—in the postwar period, our free society is supplanted by a regime of regimentation. That can happen if we have too much unemployment for too long after peace comes. It is lack of jobs on a large scale that gives dictators their opportunity . . . If collectivism comes to America it will come by default on the part of good citizens rather than design on the part of revolutionaries."

Lesson for Medicine

There is a lesson for medicine in the attitude and objectives of these qualified representatives of big business.

Medical care in the United States long since passed the point where care of the sick was the exclusive responsibility of the doctor, the patient and the patient's family. It is now definitely recognized that government has a responsibility, also, for its indigent and unfortunate, and for certain types of chronic illness. Pushing that responsibility into the accepted domain of private practice will involve relatively few mental adjustments for the average citizen provided the need can be shown to him. The answer to the problem of medicine, as to the problems of private business, is preparation, now, for the peace. It will not serve either medicine or business to cling to imponderables or cite the records of the

past. If there are gaps in medical service anywhere, if there are injustices or inequalities or scarcities, plans must be shaped now to meet them within the operation of private medicine and in coöperation with official agencies.

There Is Still Time

There are solutions for all such problems without upsetting our plan of medical practice. Since it is quite unlikely that any radical social legislation, including the Wagner-Murray-Dingell bill, will get through Congress this year or next, there is still time—but not very much time—to find those solutions.

Physicians in Minnesota have laid solid foundations for coöperation with government agencies and without sacrifice of medical control over standards of service and conditions of work. The pattern can be extended as needed. But the needs elsewhere must be assessed also and practical measures taken to meet them or the initiative will be forever taken out of the doctor's hands.

THREAT TO FREEDOM

The insurance companies are naturally opposed to the Wagner-Murray-Dingell bill. Nevertheless, certain observations made by Mr. C. O. Pauley, president of the Insurance Economics Society of America have such cogency that they should be considered by all thoughtful persons regardless of their own personal reaction to cradle-to-the-grave protections against the jolts of life.

Excerpts from an address by Mr. Pauley as published in a recent publication of the society are reprinted below for the benefit of physicians who may forget that the threat to medicine is only one phase of the over-all danger to American freedom and progress.

"Let me make it clear at the outset," says Mr. Pauley, "that any views I express with regard to compulsory government insurance are not based upon any unfavorable effect that it might have on my business. If compulsory insurance is a good thing for the American people it would be just as foolish for private insurance to oppose it as it would for employers and labor to oppose labor-saving machines. No one business can or should stand in the way of the welfare of the whole people. . .

Other Obligations

"The nation, like an individual, has just so much money to spend, all of which must come from the labors and enterprise of its people. If we want a social security program of this magnitude more than we want other things, perhaps we can have it. In considering this program, however, we must bear in mind that there are certain other obligations which come first. When this war is over we shall probably have a national debt of 300 billion dollars or more. If we assume the interest on this debt is only 2 per cent and it is amortized over a period of 50 years, it will take 9.5 billion a year in taxes. We must finance the rehabilitation of most of the world after the war, and for a time must feed and clothe millions outside our own borders. Recently there appeared in the newspapers a report of the Investment Bankers' Association in which it was estimated that our own industrial development would require 5 billions a year the first three years after the war. Our people want a constantly improved and extended system of education, we want parks and recreational facilities, we want a great many things which are not productive and in addition we must support the vast number of municipal, state and federal employes and costs of services we hope they will render us.

"Killing the Goose"

"You can take only so much in the way of taxes or contributions from the results of free enterprise without 'killing the goose that lays the golden egg.' Can our free economy, in addition to all the other burdens it must carry, support a compulsory government insurance scheme which will impose a 20 per cent or more tax on our payrolls and take 15 or 20 billions from the earnings of those who work and produce and redistribute it to those who do not work and who are not productive?

"If we embark on a social insurance scheme, the cost of which proves to be too great for our national economy to sustain, just one of two things will happen—either the actual benefits will be reduced by inflation which reduces the purchasing power of the dollar with all its attendant evils, or, as is more probable, the failure of free enterprise to give full employment and to sustain the social security burden will result in a demand that the government take over more and more functions now performed by private enterprise and until we have a completely socialized economy.

Effect on Character

"But the cost will not be entirely economic. . . What will be the effect upon the character and enterprise of a generation which knows that from before its birth in a government hospital until it is laid away in the grave, a benevolent governmental bureau will pay the costs of being born, the costs of its education, will supply its recreational needs, will furnish medical services and hospitalization in illness, provide an income during unemployment and sickness, and a

pension if permanently disabled by illness or by old age? What becomes of the incentive to rise out of the conditions in which the individual is born? Will it not result in the rise of only a few who are born with great inner driving power and ambition while the great mass of our people settle down on a dead level of security such as prevails in most European countries? Is it not possible that we may overreach ourselves in our efforts to give every man complete freedom from want and from fear without any responsibility on his part? It was the desire to achieve freedom from fear and want for themselves and their families which urged men on from our Eastern Coast to settle the wilderness and the prairies and made our nation what it is today. Perhaps the striving for the goal has been and is more important in the life of a nation than the goal itself. . . .

Political Costs

"Compulsory social insurance will also have its political costs. Here again I can only touch on one phase of the subject. During the last quarter of a century the federal government has been increasing its powers and functions at a rapid rate. Out of this has grown a bureaucracy which is increasing at an amazing speed. It is not wholly a product of the present administration, but its growth during the past decade has been tremendously accelerated. I think few of us realize the danger involved in this rapid growth of bureaucracy. It is not confined to Washington, but is spreading all over the country. . . In addition to all of these various governmental bureaus, each vying with the other for an increase in its importance, the amount of money it can spend, and the number of people it can employ, we have more recently developed hundreds of governmental corporations some of which are not even audited by the Treasury or any other governmental department. The American bureaucrat seems to have a peculiar genius for hiring additional employes. The OPA has 2,700 lawyers, for instance. England, which has a price control organization similar to the OPA, has managed to struggle along successfully with just 10 members of the legal profession, but our existing bureaus will be completely overshadowed by a bureau which will be necessary to administer a social security system such as is contained in the Wagner bill. It would necessitate a federal bureau representative in every city, village and hamlet who would go into every city and farm home. I do not need to point out the political implications of such a bureau upon the tender ministrations of which every individual would at one time or another during his lifetime be dependent.

But Bureaucrats Go On Forever

"I have never been much afraid we would lose our liberties to a man on horseback. At worst that would be only a passing phase, dependent upon the life and health of one man. But bureaucrats go on forever. Change of administration may impede their advance for a time but their steady progress to more and bigger and better bureaus and more bureaucrats goes steadily on. If we lose our liberties it will be to millions of federal employes who more and more are regulating our individual lives and curtailing our individual freedom.

"You may say, as they told me down in Washington at the Social Security Board, 'You don't want the Government to do anything.' I do want the Government to do everything that cannot be done by private enterprise or that it can do better than private enterprise and which will not impede or destroy private enterprise. Impelled by the economic and political pressure of the early 1930's we embarked upon a system of compulsory government insurance limited to old age annuities, benefits for surviving widows and children and unemployment compensation. These have operated only during the period of increasing employment. They have not existed long enough to prove themselves before venturing upon the much more difficult and costly fields of disability, medical care and hospitalization. I believe in social security, but I want to see it achieved by the efforts of the individual, as far as possible by his own initiative. I believe the government has a great place in such a program. Any system of social security, private or public, is based upon nearly complete employment. And the government should bend every effort to prevent periods of mass unemployment in private enterprise and to make possible at all times profitable employment in private enterprise for all but a small proportion of unemployables. We have made only a small beginning in conservation of health and the prevention of preventable diseases. The expenditures of the government for health conservation and the elimination of communicable and other preventable diseases should be greatly expanded. The governmental efforts should be directed primarily at causes of unemployment, accidents and disease and to the rehabilitation of those who have become impaired. In my opinion, the efforts of government should be addressed primarily to the underlying causes of unemployment and disability rather than placing emphasis upon paying its citizens for being unemployed or sick. It should encourage its citizens to provide against such contingencies by education, by savings, by insurance and all other means available; and should provide for those whose needs are inevitable by a judicious system of public assistance, rather than by fastening upon the whole American people a vast compulsory social insurance program administered by an ever-growing bureaucracy."

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

J. F. Du Bois, M.D., Secretary

Columbia Heights Midwife Pleads Guilty to Attempted Abortion

Re State of Minnesota vs. Hilda Helander.

On September 28, 1943, Mrs. Hilda Helander, sixty-five years of age, 4305 Fourth Street N.E., Columbia Hights, Anoka County, entered a plea of guilty in the District Court of Anoka County, to the crime of attempted abortion. Mrs. Helander, a licensed midwife,

was sentenced by the Hon. Leonard Keyes, Judge of the District Court, to a term of not to exceed two years in the Women's Reformatory at Shakopee, Minnesota. Because of the defendant's age and the condition of her health, the Court placed the defendant on probation for a period of two years. The defendant has a married daughter who lives on a farm near Outlook, Montana, and she stated to the Court that she desired an opportunity to permanently reside with her daughter. This arrangement was approved by both the County Attorney of Anoka County and legal counsel for the Medical Board. The defendant was also required to surrender her midwifery license for cancellation.

Mrs. Helander was arrested on July 11, 1943, following the admission of a nineteen-year-old Minneapolis girl to Deaconess Hospital. The girl, who was critically ill, while an unwilling witness, finally disclosed that she had gone to Mrs. Helander on July 8, 1943, for a criminal abortion. The amount of $25.00 was paid to Mrs. Helander. The girl ultimately recovered.

The Minnesota State Board of Medical Examiners wishes to acknowledge the splendid coöperation received in this case from Deaconess Hospital, the attending physician, Mr. Charles P. LeRicheux, County Attorney of Anoka County, and Deputy Sheriff L. A. (Mike) Auspos of Anoka County. Splendid assistance was also given by members of the Women's Bureau of the Minneapolis Police Department, notwithstanding the fact that the defendant resided just outside the Minneapolis city limits.

Minneapolis Pharmacist Pleads Guilty to Illegal Sale of Nembutal

Re: State of Minnesota vs. Casmer A. Sathre.

On October 5, 1943, Casmer A. Sathre, forty-two years of age, entered a plea of guilty in the District Court of Hennepin County, to an information charging him with the illegal sale of nembutal. Following a statement of the facts to the Court, the Hon. Edmund A. Montgomery, Judge of the District Court, sentenced the defendant to pay a fine of $200, or to serve one year in the Minneapolis Workhouse. The defendant elected to pay the fine.

Mr. Sathre, who is a registered pharmacist and operates his own store at 1825 E. Lake Street, Minneapolis, was arrested following a joint investigation made by the authorities of Anoka County and Hennepin County, the Minneapolis Police Department and the Minnesota State Board of Medical Examiners into the death of a thirty-nine-year-old Minneapolis woman. The deceased was found dead at the home of a daughter in Anoka on August 22, 1943, under circumstances indicating an overdose of nembutal. While the defendant denied having sold any of the barbiturates to the deceased immediately prior to her death, he admitted that he had refilled, on three or four occasions, without authorization, a prescription written by a Minneapolis physician in February, 1942, for the deceased.

This is the first prosecution under the 1939 State Law restricting the sale of the various barbital preparations to the written prescriptions of one authorized to issue the same, or to an authorized refill of such a prescription. The law was sponsored by Senator Harry L. Wahlstrand of Willmar, Minnesota, and had the endorsement of the Minnesota State Medical Association. The tragic death in the instant case illustrates the danger of the promiscuous sale and use of these drugs. The penalty imposed by the Court should serve as a warning that there will be no financial gain to those who violate this law. The maximum penalty is a fine of $1,000 or one year in jail. Splendid coöperation was received in this case from Mr. Charles P. LeRicheux, County Attorney of Anoka County, Mr. A. C. Horejs, Coroner of Anoka County, Captain James Mullen of the Minneapolis Police Department, Dr. Russell R. Heim, Coroner of Hennepin County, Mr. Michael J. Dillon, County Attorney of Hennepin County, and Mr. Otto Morck, First Assistant County Attorney of Hennepin County.

UNCLE SAM DRAFTS SUPPLY OF OIL OF PEPPERMINT

The entire supply of oil distilled from the peppermint plant has been ordered set aside for government action to secure a just distribution.

Oil of peppermint is important in both food preparations and pharmaceutical uses, particularly for menthol production. Most of the menthol used in the states was formerly imported from Japan.

American farmers since the war have attempted to increase the production of oil of peppermint to meet growing civilian, military and Lend-Lease requirements. Most of the United States supply is produced in Michigan, Indiana, Ohio, California and Oregon. Unfavorable weather conditions this year caused domestic production to fall short of the goal.—*Science News Letter,* October 23, 1943

CLINICAL-PATHOLOGICAL CONFERENCE

(Continued from Page 991)

media, as found in syphilitic aortitis. The wall of the aneurism is fibrous and thin. The thrombus in the aneurism is hyaline but not organized. The lungs show severe pneumonia. This is one instance where a Mexican has a chronic cough and weight loss due to something other than tuberculosis.

DR. HERTZOG: The large thrombus present in this aneurism explains why it did not pulsate. Thrombosis in an aneurismal sac is a protective phenomenon and helps to prevent rupture. This man died of bronchopneumonia. Dr. Rudolph Matas in New Orleans many years ago attempted to produce thrombosis in aneurysmal sacs by introducing coiled gold wire into the sac. One difficulty was that the wire would not always remain in the sac. The opening in this saccular aneurism was extremely small. Coughing is a common symptom in aneurisms of the arch of the aorta due to stretching of the left laryngeal nerve. Syphilis causes nearly all aneurisms of the arch of the aorta. However, it should be remembered that arteriosclerotic dissecting aneurisms may involve the arch of the aorta and the majority of aneurisms of the abdominal aorta are on the basis of arteriosclerosis, rather than syphilis.

Anatomical Diagnosis: (1) Aneurism of arch of aorta; (2) syphilitic aortitis; (3) bilateral bronchopneumonia.

INDUSTRIAL HEALTH

STANDING ORDERS FOR NURSES IN INDUSTRY

For some time the medical and nursing professions have been concerned about the employment of nurses in industry without adequate medical supervision. The Council on Industrial Health has therefore been requested to formulate standing orders for industrial nurses which can be adapted to meet the requirements of individual industrial medical departments. If no responsible industrial medical authority exists, it is recommended that the nurse request helpful instruction in this regard from the committee on industrial health of the appropriate county or state medical society.

General Relationships

Standing orders represent a preliminary understanding between physician and assisting personnel about routine conduct of a medical service. In establishing such orders in an industrial medical department, several considerations need to be borne in mind:

1. The greater the amount of personal supervision exercised by the physician directly in the industrial environment, the better is the industrial health service.
2. Standing orders cannot be written to meet every situation likely to arise in industry. They must be modified to meet specific requirements and in accordance with the training and professional competence of the assisting personnel. They should be signed by the supervising medical authority and posted prominently in the medical department.
3. The nurse in industry should assume no responsibility for service outside the field of her professional training. This applies particularly to individual case management, from which the nurse should rigidly abstain except:
   (*a*) In emergencies demanding immediate independent judgment and action.
   (*b*) Procedures of preliminary or first aid nature routinely required by reason of the nature of the work and which are clearly stipulated in the standing orders.

This statement confines itself mainly to these last named aspects of medico-nursing relations in industry. Additional reports on other functions of industrial nurses will follow as needed.

Emergency Procedure in Industry

General principles which operate in all emergency situations apply to industry as well. They are:

1. Call a physician immediately.
2. Stop bleeding.
3. Restore breathing.
4. Prevent shock and infection.
5. Do no more than is actually needed.

The supervising physician should assure himself that these instructions are thoroughly understood and should institute special training when necessary. Nurses in industry should qualify as first aid instructors.

Emergency Supplies.—Emergency packs with essential sterile supplies should be available at all times in the medical department and in first aid kits suitably located throuhout the plant. Regular inspection is necessary.

Hemorrhage—Bleeding calls for immediate attention. The nurse should notify the physician and, until he arrives, proceed as follows:

1. Expose the wound.
2. Remove obvious foreign matter.
3. Apply pressure.

Direct manual or bandage pressure firmly applied over sterile gauze packing at the bleeding site will effectively control moderate hemorrhage. Indirect compression is indicated in excessive bleeding not controllable by direct methods. Digital compression over the vessel against underlying structures either adjacent to the wound or at the nearest pressure point will usually suffice until the physician arrives. Indirect pressure should be applied proximal or distal to the wound, in keeping with the arterial or venous character of the bleeding. Hemostats or clamps should be applied whenever the emergency warrants it.

Avoid applying a tourniquet if possible. If severe bleeding in an extremity suggests the use of a tourniquet, apply a blood pressure cuff.

The nurse should remember that:

1. A direct pressure bandage should not act as a tourniquet.
2. A tourniquet must be periodically released at least every fifteen minutes.
3. No dressing should be applied over a tourniquet.
4. Asepsis must be observed at all times.

Asphyxia.—Cessation of breathing from any cause demands:

1. Artificial respiration *at once* and *at the site of the accident.*
2. Notification of the physician.
3. Maintenance of body warmth. Avoid excessive heating.

All industrial nurses should demonstrate ability to apply artificial respiration by the prone pressure method and should realize the need for its continuous application until breathing is restored or until careful repeated medical examination advises otherwise.

Shock.—Early and adequate shock treatment is life saving. Do not delay.

Common symptoms of shock following injury are pallor, perspiration and rapid thready pulse. Emergency management by the nurse should include:

1. Notification of the physician.
2. Removal of cause. If shock is due to hemorrhage, control it. If it is due to trauma not associated with bleeding, all active treatment of injury should be deferred until shock management has been in-

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stituted. Wounds should be covered with sterile dressings to prevent infection.

3. Relief of pain: ⅙ to ¼ grain (0.010 to 0.016 Gm.) of morphine sulfate, repeated if necessary, or barbiturates as routinely ordered except in injuries to the head or trunk.
4. Keeping the patient warm, dry, and on his back with his head low. Avoid overheating.

Routine Nursing Care of Injuries

Successful medical management of industrial injuries depends on:

1. Prompt treatment.
2. Meticulous cleansing and dressing.
3. Examination of deep as well as of superficial structures.

To accomplish these aims the routine functions of the nurse should be confined to care of minor wounds as follows:

1. Protect wound with sterile gauze while adjacent area is cleansed with soap and water or solvent.
2. Discard protective dressing and clean wound margins.
3. Irrigate wound with sterile water or isotonic solution of sodium chloride.
4. Apply antiseptic of physician's choice.
5. Apply dry sterile dressing, interfering as little as possible with function. Sterile dressings should be covered with protective material for use at work. The worker should be instructed not to remove the dressing but to return to the medical department if it becomes loosened or uncomfortable.

The nurse should do no more than is actually needed. The following conditions require direct medical supervision:

1. Wounds requiring débridement.
2. Those with obvious or suspected involvement of deep structures.
3. Wounds with edges which do not approximate.
4. Wounds about the head and face.
5. Contaminated wounds requiring tetanus prophylaxis.

Management of Common Injuries.—Injuries most likely to be encountered in industry include the following conditions:

1. Abrasions: Clean and apply dry dressing. Extensive or deep loss of skin, especially about the fingers and hands, needs medical attention.
2. Contusions: Treat with cold compresses directly following injury, later with moist heat. If soreness or disability persists or if deep involvement is suspected, refer to the physician.
3. Lacerations: Clean and apply dressing as directed. Any possibility of injury to joints, nerves or tendons should be brought to the physician's attention at once.
4. Puncture Wounds: Puncture wounds through the skin need direct medical supervision to avoid or treat severe infection. If superficial, clean and apply sterile dressing.
5. Slivers and Splinters: Penetration through the skin by slivers or splinters always carries the risk of an infected puncture wound and should be treated as such. Those lodged superficially and easily removed without added trauma or incision may be extracted aseptically by the nurse.
6. Burns and Scalds: Clean minor burns with soap and water. Apply petrolatum or 5 per cent boric acid ointment, bandaging firmly without interfering with function. Leave blisters alone.

   In all other cases:

   (*a*) Notify the physician.
   (*b*) Cover the burned area with a sterile dressing or sheet moistened with isotonic solution of sodium chloride or 5 per cent sodium bicarbonate solution.
   (*c*) Combat pain and shock.

In the absence of specific orders, chemical burns should be treated by irrigation or immersion in water for at least twenty minutes and then by dressing.

7. Sprains and Strains: Treat first with cold compresses, elevation of the part and rest. A physician's advice is necessary regarding strapping, other methods of support or fixation, further examination or special therapy.

Fractures

Preliminary steps for the nurse are:

1. Call a physician at once.
2. Keep the patient quiet and warm.
3. Immobilize before any movement is atttempted.
4. Do not attempt reduction.
5. If the fracture is compounded, cover the site of the fracture with a dry sterile dressing. *Do not cleanse or reduce.*

Special instruction in splinting should be provided every industrial nurse.

Eye Injuries

Rigid asceptic technique must be scrupulously observed in all eye conditions. Never attend consecutive patients without sterilization of instruments and careful hand washing. Remember that early symptoms of infection simulate foreign body.

Minor Burns.—Do not apply ointments to minor burns of the skin about the eye. Apply a sterile dressing and refer to the physician.

Burns of the Eye.—1. Chemical Burns: Irrigate chemical burns of the eye *copiously* and *at once* with water, preferably by immersion. Neutralizing solutions are usually inadequate or unavailable. The rapidity with which the irrigation occurs is more important than the type of solution used. Continue to irrigate at least twenty minutes by the clock.

2. Hot Metal Burns: Aply a sterile pad and refer at once to the physician. Do not irrigate. An anesthetic should be aplied as ordered by the doctor.

Every burn of the eye should receive competent medical attention early.

Foreign Bodies.—The nurse should attempt to remove only those foreign bodies of the eye which can be readily located and which can be easily washed out or removed with a dry sterile cotton applicator. An antiseptic may be applied if the physician so orders.

Direct medical care is essential:

1. If the foreign body cannot readily be located. Stains to aid in the location of foreign bodies should be used only on specific medical order.
2. If removal requires any instrumentation.
3. If irritation or pain persists after removal.

No person with an eye injury should be discharged without examination by a physician.

"Flash" Injury.—First aid treatment should include:

1. Local anesthetic as ordered.
2. Cold compresses.
3. Sedatives.

Persistent pain following flash needs medical examination and treatment.

Conjunctivitis.—Conjunctivitis or other forms of conjunctival irritation should be referred routinely to the physician or ophthalmologist.

Head Injuries

Until the physician takes over, the nurse should:

1. Keep patient lying down.
2. Elevate the head.
3. Apply ice cap or cold compress. *No sedatives.*
4. Record pulse and respiration every ten minutes.

Clip or shave and cleanse areas adjacent to scalp lacerations, and cover with a sterile pad.

Chest and Abdominal Injuries

Contusions of the chest and abdomen with or without external evidence of injury may result in trauma to underlying organs.

Until seen by the physician, such patients must be:

1. Kept warm and quiet.
2. Allowed no sedatives.
3. Have pulse, temperature and respiration recorded frequently.
4. Suitably bandaged to avoid contamination.

In case of abdominal injury give nothing by mouth.

Nonoccupational Illness

Treatment of injury or illness which has no relation to occupation is not a function of the industrial medical department except:

1. First aid for emergency sickness. Such measures as the situation demands must be taken until notification of the family physician discharges responsibility.
2. For minor ailments which temporarily interfere with an employe's comfort or ability to complete a shift and for the relief of which a physician would not ordinarily be consulted.

In all relationships of this kind, judgment and tact are required of the industrial nurse. Several principles apply:

1. Before giving any treatment, the temperature, pulse, general appearance and a history of the presenting complaint should be recorded.
2. Palliative treatment, especially for chronic or recurring disorders, should not be repeated.

Every properly trained nurse understands the difference between attention of this kind and systematic treatment.

Care of Minor Illness and Symptoms

Persistent or augmenting symptoms of irritation, discomfort or disability suggest faulty work environment. The nurse should not hesitate to ask for medical examination of workers and of the premises.

Fever.—A rise in temperature of 1 degree suggests medical consultation before work is resumed. Findings should be checked by repeated thermometer recordings.

Headache.—Record temperature. If headache is accompanied by dizziness, nausea, vomiting, stiff neck, injury, history of recurrence, fever, general malaise or other symptoms the patient needs medical attention. If not, give an analgesic as ordered by the physician.

Remember that headache or dizziness may be premonitory signs of intoxication.

Unconsciousness.—1. Fainting. Usual symptoms are pallor, with shallow breathing, slow and weak pulse. Period of unconsciousness is of short duration.

Keep the patient lying down with head lowered until fully recovered. Be sure the patient has plenty of fresh air. Clothing should be loosened and stimulating inhalants used, such as ammonia or smelling salts.

2. Other causes. If other signs are present or if unconsciousness persists longer than a few minutes, call for medical assistance. *Give nothing by mouth.*

Toothache.—If there is a cavity, the nurse may pack it with cotton dipped in oil of cloves for temporary relief. For further examination and treatment refer to a dentist.

Nosebleed.—Spontaneous nosebleed may be treated by cold packs or pinching the sides of the nose against the septum. Keep the patient sitting erect or standing and loosen the collar if it tends to constrict the neck. Advise the patient not to breathe or blow through the nose for an hour or two after bleeding has stopped.

Bear in mind that certain occupational exposures are manifested by nasal damage and bleeding.

Sore Throat.—Patients with sore throat may be given a hot saline gargle if they have a normal temperature. Do not "paint" the throat. Any persistent sore throat or one associated with fever needs medical care at home.

Respiratory Irritation or Infection.—Repeated or persistent signs of bronchial or chest irritation without associated infection suggests an unfavorable occupational exposure. A plant hygiene survey is indicated.

Persons having acute respiratory infections with elevated temperature, cough, sneezing or nasal discharge should be sent home for proper segregation, rest and medical attention. In mild infections, work may be continued if under medical or nursing supervision simple measures will control symptoms and prevent spread.

Available medical evidence at the present time cannot support routine administration of cold vaccines or vitamin preparations as methods of reducing the incidence or severity of acute respiratory infections.

Frequent colds or chronic respiratory conditions require special medical consideration.

Abdominal Distress.—Early signs of occupational intoxication may be abdominal in character. In any case abdominal distress, nausea or pain, especially if severe or persistent, requires competent medical diagnosis and management.

Laxatives should never be dispensed from an industrial medical department.

Dysmenorrhea.—Painful menstruation not associated with fever or gastrointestinal disturbances may be treated with an analgesic ordered by the physician and the patient placed at rest with heat to the lower part of the abdomen. If there is no relief or if other signs or symptoms present themselves, she should be referred to her physician.

Patients with recurrent severe dysmenorrhea should not be given palliative treatment. They should be referred for examination and treatment.

Dermatitis

Management of skin disorders in industry depends on cause.

Specific Irritants.—Materials or processes in the plant capable of causing skin disease should be identified and special orders provided for control. Competent dermatologic consultation is essential in all obscure or refractory situations.

Nonspecific Skin Disease.—Nonspecific skin irritation in industry is almost entirely assignable to faulty personal hygiene. The nurse can do much to improve washing routine, the use of dependable protective coverings, the wearing of clean work clothing, maintenance of satisfactory housekeeping in the plant and the general maintenance of accepted hygenic procedure.

Pregnancy

A definite policy regarding employment during pregnancy should embrace the following recommendations:

1. The employe should notify the proper authority in industry about her pregnancy within the first trimester.
2. She should obtain a statement from her own physician—
   (*a*) That her work is not contraindicated.
   (*b*) Regarding the length of time she should work.
3. Special attention should be given to the nature of the work. Pulling, pushing and lifting must be kept within safe limits. Rest periods will tend to minimize emotional and physical instability during pregnancy.

4. Ordinarily work should terminate by the thirty-second week (within six weeks of term). If contraindications arise within this period, the employment should stop.
5. Return to work is inadvisable before six weeks after delivery and then only on notification of the employer by the physician.

Equipment and Supplies

Space which can command privacy and which can be kept clean and properly prepared for emergency and routine services by the nurse should be provided in the plant. Special attention should be given to heating, light, ventilation and accessibility.

Furnishings and Supplies

General Furnishings:
1. Sink
2. Instrument cabinet
3. Sterilizer
4. Dressing table
5. Leg rest
6. Cot
7. Stretcher
8. Mirror 10 by 12 inches
9. Foot-pedal waste can
10. Waste basket
11. Storage cabinets
12. Paper towel rack
13. Adhesive rack
14. Record file
15. Scale

Instruments and Supplies:
1. Scalpels
2. Splinter forceps
3. Tissue forceps
4. Hemostatic forceps
5. Bandage scissors
6. Surgical scissors
7. Hand magnifying glass
8. Syringes
9. Assorted hypodermic needles
10. Assorted surgeons' needles
11. Needle holder
12. Assorted bandages
13. Adhesive plaster
14. Cotton
15. Applicators
16. Assorted sutures
17. Assorted splints
18. Assorted jars and basins
19. Test tubes
20. Safety razor and blades
21. Hot water bottle
22. Ice cap
23. Crutches
24. Tourniquet

Drugs: (as ordered by the physician or medical adviser)
1. A stimulant
2. An emetic
3. Analgesics and sedatives
4. Antiseptics

The accompanying check list of furnishings and supplies suitable for a small plant dispensary should be augmented by equipment for emergency treatment or other special medical requirements as ordered by the plant physician or other medical adviser.

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(*b*) Plant Hygiene Studies.
(*c*) Outline of Procedure for Physicians in Industry.
(*d*) Women in Industry.
(*e*) Control of Common Respiratory Infections.
(*f*) Indiscriminate Administration of Vitamins to Workers in Industry.
(*g*) Recognition and Prevention of Industrial Dermatitis.

MENINGITIS STOPPED

Meningitis epidemics can be stopped almost immediately by prophylactic use of sulfadiazine, Col. Dwight M. Kuhns and Capt. Harry A. Feldman, of the Army Medical Corps, reported at the wartime conference of the American Public Health Association, from their experience in the Fourth Service Command.

Chief difficulty heretofore in stopping a meningitis epidemic has been that, although the patients were isolated, it was impractical if not impossible to find and isolate all the healthy carriers of the germs.

Giving small doses of sulfadiazine to all the personnel of a single unit at the same time immediately after the first cases signal the approach of an epidemic will immediately eliminate all the carriers and at the same time reduce the attack rate, or new cases, almost to zero, the Army medical officers found.

Careful laboratory methods for detecting the carriers and identifying the type of meningococcus they carry are important, they emphasized. The type of germ predominating is a forecast of the extent and rapidity of the impending epidemic.

The laboratory and prophylactic methods worked out for the Army, they state, can be applied equally well to certain civilan groups, such as schools, orphanages, asylums, camps and the like.—*Science News Letter*, October 23, 1943.

STATISTICS SHOW THAT CANCER IS CURABLE

Too many people still look on cancer as an absolutely hopeless condition. Dr. Ira T. Nathanson, of Harvard Medical School, challenges this gloomy view. (*New England Journal of Medicine*, Sept. 16.)

"It is a well-accepted fact," he declares, "that present-day treatment is effective in the early stages of the disease, before it has spread to distant organs or tissues that are essential to life. Moreover, the prognosis is far from hopeless, even in more advanced cases."

In several forms of cancer, he adds, "the rate of cure is higher than that of some other diseases."

The chances for any given patient being cured of cancer depend on many factors, such as the location of the cancer, the type of cancer, and how early in the disease treatment is started.

In cancer of the skin, for example, the curability rate is 48 to 68 per cent. In cancer of the breast the relative curability rate, based on results five years after operation, ranges from 30 to 51 per cent. The absolute curability rate, for all patients who have treatment of any kind including that which is merely palliative is 22 to 28 per cent. In cancer of the stomach, the five-year curability rate of all patients who enter the hospital is 5 per cent. When operation is possible, however, and it is in about half the patients with stomach cancer, the outlook is much brighter, with survival rates of from 15 to 50 per cent.

Even when cancer is discovered too late for an absolute cure to be achieved, suitable treatment can often give the patient many months or even a few years more of comfortable life. No one who thinks he has cancer should feel too discouraged or frightened to see a doctor. The condition may not even be cancer, and if it is, the outlook may be much brighter than he thinks.—*Science News Letter*, October 23, 1943.

Minnesota Academy of Medicine

Meeting of May 12, 1943

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Minneapolis Club on Wednesday evening, May 12, 1943. Dinner was served at 7 o'clock and the meeting was called to order at 8:15 by the President, Dr. H. B. Zimmermann.

There were forty-nine members present.

Minutes of the April meeting were read and approved.

The scientific program followed.

Dr. Wallace Cole, of Saint Paul, gave a lantern slide talk on some orthopedic cases.

ENTEROGENOUS CYSTS OF THE RECTUM

HAROLD E. HULLSIEK, M.D.

Saint Paul, Minnesota

Enterogenous or enteric cysts occur not infrequently at many points along the intestinal tract, but a search of the literature for the past ten years reveals but few located in the rectum. McLanahan and Stone, in *Surgery, Gynecology and Obstetrics,* say, "Enteric cysts . . . are of peculiar interest not only because of their rarity but also because of the problem they present in diagnosis and the speculation they stimulate as to their origin." They refer to them as unusually rare tumors and report two cases, one in an infant and one in a man sixty-eight years old. Evans, in an extremely comprehensive article in the *British Journal of Surgery* describes cases in which cysts occurred in the esophagus, stomach, duodenum, jejunum, ileum, ileocecal region, vitello-intestinal tract, and sigmoid, but none in the rectum.

He thinks that epithelial misplacements in the intestinal tract, either cysts or diverticula, originate in the diverticula found in the developing embryo. I believe another factor entering into the formation of anorectal cysts is the presence of the deep epithelium-lined tracts starting in the columns of Morgagni. Lately considerable investigation of these structures has been carried out, the results of which make it evident that these ducts are the etiologic factor in many peri-anal abscesses, fistulæ, and blind internal sinuses. There seems to be no valid reason why they might not, in the absence of inflammation, produce cysts.

Considered histologically, enteric cysts usually show the same lining structure as that of the normal bowel with which they are contiguous, but may be found to exhibit every known variation in the mucosal layer. In some, the changes in the lining of the cyst cavities are explained by intracystic tension or inflammation. Ewing says "The cavity is usually single and the contents are mucinous, colorless, yellowish, or brownish fluid. The wall resembles that of the intestines and may contain smooth muscle, mucosa, crypts, lymphoid tissue and a lining of cylindrical, stratified or cuboidal epithelium." The commonest sites are the ileum at the point of Meckel's diverticulum, in the mesentery, or near the umbilicus. They may be submucosal, intermuscular, subperitoneal, or intermesenteric.

The symptoms are governed by the size of the cyst, its location, its encroachment on other organs, and the presence or absence of inflammatory change. In my patients, the cysts were easily felt with the finger as extra-rectal masses, fairly firm, tensely fluctuating in two cases, and varying in size from that of a hazelnut to a walnut. The absence of severe pain together with a relatively low leukocyte count make easy the differentiation from pari-rectal abscess. These masses do not feel like new-growths, and on proctoscopic examination the absence of any change in the mucosa may be noted. I should like to report four cases.

Case 1.—Miss J. L. is a white, unmarried woman, aged thirty-three. For the past two months she has felt a sensation of pressure in the rectum, together with an inability to completely empty the rectum. There was no actual pain and no bleeding. On digital examination a mass was felt in the right anterior quadrant of the rectum immediately above the sphincter, and one in the right posterior quadrant. These were about the size of hazelnuts, fairly firm, and it was thought, fluctuating slightly. Except for a slight reddening over one cyst, there was no change in the mucosa. The leukocyte count was 12,500.

Under sacral anesthesia the anal canal was relaxed and the tumors visualized. They were estimated to be respectively 2 cm. and 1 cm. in diameter. The one on the left side was grasped by a forceps and partially prolapsed. It was incised and a cloudy, gelatinous substance escaped. The same procedure was followed on the second mass. The cysts showed definite cavities, the edges of which were widely trimmed off in an effort to convert the cavities, into continuous portions of the rectal wall. They were packed temporarily with iodoform gauze. The patient left the hospital on the sixth day, and shortly afterward was discharged, examination having shown the cavities practically obliterated.

Case 2.—Mr. H. W. is a white man, aged thirty-four. He complained of pain and tenderness in the anal canal, which was associated with slight showing of fresh blood, with an exaggeration of the symptoms each time the bowels moved. Examination showed a deep, chronic anal ulcer at the right of the posterior commissure, which explained the pain at stool and bleeding. Digital examination, however, revealed a tense and slightly tender mass beneath the mucosa of the posterior rectal wall. His leukocyte count was 11,000. A diagnosis was made of an anal fissure, and of a probable cyst of the rectum.

He was operated upon under sacral anesthesia, at which time a deep ulcer was dissected from the pos-

terior commissure, and a partial proctotomy done. The mass in the rectal wall was incised and contained a glairy, mucinous material. The edges of the cyst were removed and the cavity packed. He was discharged from the hospital on the fifth day. He was seen in the office every three or four days and shortly it was seen that the cavity was pocketing, and not flattening out to become a part of the rectal wall. It had too small an opening and was filling up with debris and not emptying properly. He was readmitted to the hospital twenty-five days after his first admittance, and again under sacral anesthesia, was operated on. This time the edges were trimmed more widely and the cavity packed as before. He left the hospital in three days and his convalescence from then on was uneventful. He has since completely recovered.

Case 3.—Miss P. B. is a white, unmarried woman, aged sixty. She states that for the past two months she has had a sensation of weight and slight aching in the rectum. The bowels are regular and there has been no bleeding. Examination shows a firm mass the size of a walnut beneath the mucosa on the left side of the rectum. It is not movable, and is only tender on very firm pressure. Her leukocyte count was 9,650. Diagnosis was made of a rectal cyst.

She was operated upon under sacral anesthesia, the mass found to be a cyst filled with cloudy gelatinous material. The cavity was marsupialized and packed. The pathological report on the tissue removed reads as follows: Section shows a fibromuscular tissue which is edematous and infiltrated with plasma cells and leukocytes. Diagnosis: Wall of a chronic abscess. This illustrates Ewing's statement that many changes found in the linings of the cysts can be accounted for by intracystic tension or inflammation. The histological picture here is one of inflammation, and yet this was very evidently not primarily an abscess. This patient was discharged on the eighth day, and made an uneventful recovery.

Case 4.—Mrs. M. M. is a white, married woman, aged thirty-five. Two and a half years ago she had what she thinks was a rectal abscess opened through the rectum. Six months ago she had the same procedure done, both operations having been performed in another city. For the past two weeks she has had rectal pain, not severe, and not associated with bowel movements, together with a sense of fullness in the rectum. There has been no rectal bleeding. Examination showed an indurated, slightly tender mass in the rectal wall on the left side. Anuscopic examination shows no change in, or involvement of, the rectal mucosa. There is no external tenderness on deep pressure. Her leukocyte count is 15,000. A diagnosis was made of probable cyst of the rectum.

She was operated upon under sacral anesthesia, the tumor mass on the left side being incised, with the escape of a clear mucinous substance. The cyst cavity was treated as were the former ones, and she left the hospital on the eighth day. Her recovery was uneventful.

Summary

Enteric cysts may occur at any point along the intestinal tract, but are apparently relatively rare in the rectal area. Their origin is assumed to be in small diverticula formed during fetal life, some of which later close off completely to form cysts. Histologically their linings resemble the elements of the adjacent bowel, but are capable of many variations.

The diagnosis is not difficult and the cyst, when found, should be treated surgically. In my cases they were treated from within the rectum, but may, especially if the cyst is large and accessible, be removed through a skin incision.

References

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2. Evans, A.: Developmental enterogenous cysts and diverticula. Brit. J. Surg., 17:34-83, 1929.
3. Ewing, James: Neoplastic Diseases. p. 1030. Philadelphia and London: W. B. Saunders Co., 1931.
4. McLanahan, Samuel and Stone, Horvey B.: Enterogenous cysts. Surg., Gynec. and Obst., 58:1029, 1934.
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Discussion

DR. ARNOLD SCHWYZER, Saint Paul: The question is —How do these cysts happen? How can the epithelium be outside of the rectal wall?

Quite a number of years ago, Tavel, an excellent Swiss surgeon, wrote a paper on this subject and he showed that the ordinary Lieberkuhn glands in the colon and sigmoid become longer in the rectum and when you get down toward the lowest portion of the rectum they become very long and deep. He showed sections where they go not only all through the bowel wall but into and through the internal sphincter. That must be the origin of these cysts. The cysts are simple in appearance and we must consider them as something like retention cysts from the abnormally deep-seated fundi of these lower rectal glands.

DR. HULLSIEK, in closing: I am fairly certain that these cysts do not occur frequently—what Dr. Schwyzer says is quite true. The ducts that start in the crypts of Morgagni may be very deep and may form sinuses, cysts, or abscesses. There may be multiple small cysts, but those in my cases were producing symptoms and were evident on examination. I know I have not seen others since every mass I have encountered in the rectum has been explored. The type mentioned by Dr. Zimmermann was further up the rectum and very likely developmental in origin.

If the cysts are not large, it seems to me easier to operate on them intrarectally. In large ones, if accessible, one may have to take out the coccyx and remove the cyst through a skin incision. One of my patients I saw in 1928; the other three have occurred in the last two years. These are the only ones I have encountered in many hundreds of rectal examinations.

ACUTE OBSTRUCTION OF THE BOWEL

A. E. BENJAMIN, M.D.

Minneapolis, Minnesota

I desire to report a case of acute obstruction of the bowel. Obstruction of the bowel is much easier to diagnose now than it was twenty to thirty years ago. There is little excuse for a patient dying from obstruction of the bowel nowadays. When I first began practice these cases were less easily handled.

The case I'm reporting tonight is a single man, sixty-three years of age, a farmer. His case is interesting because of the various troubles and complications he has had. When I saw him first in 1938, he suffered with an incisional hernia from an old appendectomy with drainage. He also had a right inguinal hernia, hemorrhoids and a right-sided hydrocele.

This patient was operated on elsewhere for a ruptured appendix in 1925 and following this he had suffered more or less with constipation and abdominal pains in the lower right side and had gradually developed a large incisional hernia. He occasionally had symptoms of acute obstruction.

Just previous to the time I saw him first, these obstruction symptoms were quite pronounced. Upon ex-

amination I found a large protruding incisional hernia which caused much pain. He wore a bandage which gave him some relief. The inguinal hernia, hydrocele and hemorrhoids caused additional discomfort. The hemorrhoids bled occasionally and the mucous membrane of the bowel prolapsed some. As the incisional hernia was the most distressing condition, we operated by taking out a portion of the skin over the hernial area. This skin was very thin and fixed to the underlying muscle. The gap in the abdominal wall was about 3 by 5 inches. The incision was extended downward and outward and the skin dissected from the fascia. A flap of fascia at the edge of the wound was dissected loose and turned upward over the defect in the abdominal wall. The underlying thin muscle was imbricated. The abdominal cavity was not opened. The fascia was stitched firmly so as to leave no dead spaces. This gave very good support and still left sufficient fascia and muscle for support below. The patient was troubled with some nausea and vomiting, so nasal suction was instituted.

While in the hospital we decided to repair the inguinal hernia and operate on the hydrocele, this we did two weeks after the incisional hernia operation, with a local anesthetic. The patient remained in the hospital about fifty-two days. Off and on he had some distress with his bowels and felt nauseated but finally recovered, felt well and went home.

In March, 1943, the patient returned having had some severe attacks of nausea, vomiting and pain. A partial obstruction apparently existed. He was operated on March 20, 1943, at which time we found omentum and intestines were quite badly adhered, especially at the old appendicial scar. On account of the intimate union of the parietal and visceral peritoneum, it was difficult to separate the bowel from the abdominal wall. The intestine was accidentally opened during the procedure but the rent was repaired at once. All disabling adhesions were corrected and the abdomen was closed. Nasal suction was used and a limited diet was continued for some time.

Finally, we decided to operate on the hemorrhoids. This was done about two weeks after the laparotomy. He made satisfactory progress for several days, when he began to have pain, some distention and nausea. Nasal suction was instituted. After three days' trial without definite improvement an operation was decided upon, especially as the x-rays confirmed our opinion.

A left-sided incision was made and two loops of small intestine, each twisted in the form of a volvulus and bands were found constricting the bowel. These were separated and the bowel untwisted. There were no adhesions to the abdominal wall where the previous operation had been made but a loop of small intestine was adherent to the abdominal wall near the hepatic flexure. This could only be corrected by a second incision over that point. This was made through the right rectus, the bowel separated and the abdomen closed.

After a rather stormy time, with the use of nasal suction and saline and glucose intravenously, the patient recovered, the bowels acted well and he felt well. There was a little sloughing of the fascia on the right side on account of imperfect circulation. The wound finally healed and the patient went home happy.

The meeting adjourned.

E. V. Kenefick, M.D.,
Secretary.

In Memoriam

LOUIS J. COOKE

With the death of Dr. L. J. Cooke, or "Doc" Cooke, as he was affectionately known by thousands of students and alumni, the University of Minnesota has lost its number one athletic personality. It is probable that more former students of the University found that they had more in common in Doctor Cooke than they had in any other person or thing connected with the University. Every student at the University for forty odd years knew him either directly or indirectly either as a coach, lecturer, toastmaster, administrator, medical examiner, all round good fellow, or as a competitor in some form of sport in which he was active. He left his mark on Minnesota athletics as no other person ever will. All minor sports at the University (except golf and tennis) along with intramural athletics were introduced there by him and then kept alive during their lean years until they attained full intercollegiate recognition. These included gymnastics, wrestling, fencing, hockey and swimming. Although some basketball was played at the University before he became attached to it, it was only when he took the sport over and became the coach that it became an organized activity. He installed a system of gymnastic work at the University including his now famous lecture course in Personal Hygiene. He promoted very successfully three great circuses at the University as a part of his physical program. At one time or another he served the University in one of the following capacities: Gymnasium Director, Medical Examiner, Basketball Coach, Baseball Coach, Minor Sports Coach, Director of Ticket Sales, President of the Western Intercollegiate Gymnastic Association, President and Co-founder of the Northwestern Gymnastic Association, Member of the Collegiate Basketball Rules Committee and Medical Director for the Student Army Training Corps during the last war. He was a competitor of no mean ability in baseball, basketball, handball, tennis and squash. Many a senior can remember Doc's curves and change of pace when he pitched in the traditional Senior-Faculty baseball game at commencement. He was equally at home afield with a fowling piece and could bring home his limit along with the rest of the boys. His whole outlook on life was competitive and he had an unconquerable desire to win, no matter what the issue was, whether tennis, getting excused from gym, or those tickets you wanted on the fifty yard line. Sooner or later you felt his great desire to be the winner of the bout. There were three great things in his life and they completely occupied and absorbed him—his job, his family and his church. He had no other interests. He always delivered the goods and more on his job. He was never late at home for dinner and could always be depended on to take the part of the old maid in the church play. There were never any activities in any of these fields that did not find him a stand-out leader and performer.

Louis J. Cooke was born in Toledo, Ohio, on February 15, 1868, and died at his home in Minneapolis on August 19, 1943. He attended public schools in Toledo and then went to the Y.M.C.A. Training School at Springfield, Massachusetts. He then attended the University of Vermont and matriculated in Medicine. During this time he was director of the local Y.M.C.A. physical activities and was a star pitcher on the Vermont star baseball team. He got his degree in medicine in 1894 and then left college to take up his career as a physical educator. He served as Y.M.C.A. physical director at Toledo, Ohio, Duluth, Minneapolis, and St. Paul. In February, 1897, while serving as Physical Director at the Minneapolis Y.M.C.A., he was induced to come to the University and take over direction of the physical education program. He worked part time until the end of the year and then in the fall he quit the Y.M.C.A. From September, 1897, until 1913 he served as gymnasium director and medical examiner and owned and operated a private gymnasium in downtown Minneapolis. In 1913 he became director of Physical Education for men and continued in his capacity of medical examiner. In 1922 when the Department of Athletics and Physical Education for men was created he became assistant to Mr. F. H. Leuhring who was chosen to head the new department. He continued in that capacity and as director of ticket sales until his retirement in 1936.

Doctor Cooke was not only a teacher of physical education, he was a pioneer in that field, and for that pioneering, the University and the Northwest in general owe him a great debt of gratitude. He established the Department of Physical Education at the University. He pioneered in the medical examination of college students and later aided in the establishment of the University Health Service. He introduced some major sports and nearly all the minor sports at the University. He originated the Michigan Jug tradition. He kept the jug hanging over his desk for years in the old armory and finally at a Michigan pre-game banquet he presented it and suggested that it be a trophy for the two teams. It is today the nation's outstanding college trophy. He was closely identified with many secondary physical education enterprises including the Western Intercollegiate Gymnastic, Wrestling and Fencing Association, the Northwestern Gymnastic Association, the Twin City Physical Education Association, Sigma Delta Psi, an honorary athletic fraternity, and many other lesser physical education activities.

The Doctor's contribution in basketball alone entitles him to immortality at the University. He played the game from its beginning and aided in its invention. He was still an excellent forward at the time of his retirement from active work. He coached several championship teams at the University and his 1919 team went through the season without a defeat.

Although he never held the position of Director of Athletics at the University, his greatness as an athletic personality was recognized in naming the new athletic

building, Cooke Hall, after him. This was an honor that few men have accorded them while still living.

He was a member of Alpha Kappa Kappa fraternity while in college, and installed the Minnesota chapter here in 1898. He continued active in chapter affairs until the time of his death.

To have accomplished so much in one lifetime with the help and aid of all concerned would have been a great attainment, but to have done so amidst the stresses and strains of a rapidly-developing institution with all its growing pains and striving for power, makes it really a colossal achievement. He kept right side up and kept going through several great struggles, including the fight between the students and the faculty for the control of athletics. He fought in these struggles when he had to but he usually had the advantage of his opponents because he fought for a cause instead of personal power. Athletic and Physical Education in the middle west will bear the imprint of his greatness for a long, long time. His death has been a great loss to the University, to the State and to the whole middle west.

W. K. Foster, M.D.

JOHN BUTLER

Dr. John Butler died at Northwestern Hospital, Minneapolis, on September 17, 1943. He was born in Berlin, Wisconsin, and spent his early years in that vicinity. After a period of railroad employment he, in company with his brothers, spent some time in Alaska where he was interested in mining.

In 1898 Dr. Butler entered the University of Minnesota Medical School, graduating in 1903. Associated there with the late Dr. Max Vanderhorck it was natural that he should become interested in dermatology. In 1910, after study in Europe, he limited his work to dermatology and practiced in Minneapolis up to the time of his last illness.

Dr. Butler was Assistant Professor of Dermatology at the University, a member of the Hennepin County Medical Society, the Minnesota Academy of Medicine, and the Dermatological and Urological Societies. He also belonged to the Minneapolis, the Minnekahda, the Lafayette and the University Clubs.

Dr. Butler's outstanding characteristics were his great capacity for friendship, his love of life and keen enjoyment of the society of those he cared for. Among the legion of his friends he leaves a place which can never be filled.

J. E. H.

SURGEON LIEUTENANT WILLIAM LYON MACKENZIE KING

Word has been received that Surgeon Lieutenant William Lyon Mackenzie King was lost at sea when the British destroyer St. Croix was torpedoed recently in the North Atlantic. Surgeon Lieutenant King, a nephew of Prime Minister Mackenzie King of Canada, was born at Ottawa, Ontario, Canada, January 2,

1913. He received the degree of doctor of medicine from the University of Toronto in 1937 and was intern at Toronto General Hospital from July, 1937, to July, 1938. Thereafter, from July, 1938, to June, 1941, he was a fellow in surgery in the Mayo Foundation and received the degree of master of science in surgery from the University of Minnesota in June, 1941. Before he joined the Royal Canadian Navy in 1942, he was house surgeon at St. Michael's Hospital in Toronto. He is survived by his wife and two children, one of whom was born in Rochester.

CAPTAIN LESTER W. BAIRD

Word has come of the sudden death of Captain Lester W. Baird, M.C., A.U.S. Captain Baird was born at Edwardsville, Illinois, April 19, 1907. He received the degree of doctor of medicine from the University of Illinois in 1933 and was successively intern resident physician at the Santa Clara County Hospital, San Jose California. Thereafter, from 1934 to 1937, he was a fellow in radiology in the Mayo Foundation. Before he entered the army he was a member of the Scott and White Clinic, Temple, Texas. Captain Baird became chief of roentgenology at Camp Carson, Colorado, where he died of coronary occlusion on October 6.

SHORTENING CONVALESCENCE

Sick and wounded men of the Army Air Forces get well faster and go back to military duty in "top" physical condition as a result of the convalescent-rehabilitation training program in Air Forces hospitals, Brig. Gen. David N. W. Grant, the Air Surgeon, reported at the wartime conference of the American Public Health Association.

The program has been in operation for the past seven months, General Grant reported. During this time approximately 16,000,000 man-hours in physical and educational training have been given and the teaching rate at the present time is about 2,500,000 man-hours per month.

The sick soldiers are reconditioned physically by a planned and organized physical rehabilitation program. At the same time, the many hours of convalescence that are usually wasted are used for the educational program designed to disseminate knowledge and thereby make these men better soldiers. If the man cannot go back to military service, he is helped by this program to return to productive civilian life.—*Science News Letter*, October 23, 1943.

Aristotle, the ancient Greek philosopher, advised over 2,000 years ago the eating of liver to prevent *night blindness.*

REPORTS and ANNOUNCEMENTS

WABASHA COUNTY MEDICAL SOCIETY

The seventy-fifth annual meeting of the Wabasha County Medical Society was held at Plainview, Minnesota, October 7, and was marked by a good attendance. The business session in the afternoon was followed by a dinner which was attended by twenty-six members, wives and guests.

At the evening session the President's Address was presented by Acting President, Dr. D. P. Dempsey Kellogg, President T. G. Wellman being absent in military service. Dr. Kellogg's subject was "Obstetrics." Dr. B. J. Terrell, Superintendent of Buena Vista Sanatorium, Wabasha, presented a paper on "Tuberculosis and Other Chest Conditions." Dr. E. C. Bayley of Lake City, delegate to the State Medical Association, gave a review of the proceedings of the ninetieth annual session of the House of Delegates. Dr. W. F. Braasch of Rochester, secretary of the National Physician's Committee, addressed the group on "What the County Medical Society Should Do." This was followed by a short talk on subjects relating to public health and sanitation by Dr. F. M. Feldman of Rochester, representing the State Board of Health, Third District.

Entertainment for the women guests was provided at the home of Dr. and Mrs. D. G. Mahle.

Officers elected for the coming year are:

President—Dr. E. C. Bayley, Lake City

Vice President—Dr. C. G. Ochsner, Wabasha

Secretary-Treasurer—Dr. W. F. Wilson, Lake City

Delegate to State Association—Dr. E. C. Bayley, Lake City

Alternate—Dr. E. W. Ellis, Elgin

Censor for three years—Dr. W. J. Cochrane, Lake City

Censors holding over—Dr. W. H. Replogle, Wabasha, and Dr. D. G. Mahle, Plainview.

WEST CENTRAL MEDICAL SOCIETY

The West Central Medical Society held its annual meeting in Morris, October 13, 1943. At the scientific session following dinner, Dr. Howard Johnson of Ortonville read a paper on "Penicillin" and Dr. Charles Bolsta discussed the present status of medical practice and prospective socialized medicine.

Election of officers resulted as follows:

President—W. C. Rydburg, Brooten

Vice President—B. W. Jarvis, Starbuck

Secretary-Treasurer—Herman Linde, Cyrus

Delegate to MSMA—F. W. Behmler, Morris

Alternate—Charles Bolsta, Ortonville.

Of General Interest

Dr. Charles W. More of Eveleth was guest of honor at a meeting of the Range Medical Society held in Eveleth, September 28.

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According to information received from his family, Dr. J. K. Keeley, former fellow in the Mayo Foundation, is a prisoner of war in the Philippine Islands.

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Dr. Oliver M. Porter, who practiced in Minnesota before going to Sisseton, South Dakota, several years ago, will return to this state this month, having opened offices in Willmar for the practice of general medicine.

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Dr. Floyd Burns of Milan has been commissioned Lieutenant in the Medical Corps of the United States Navy and has reported for duty at the Great Lakes Naval Training Station.

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Dr. A. F. Geis of Graceville has received his call for induction in the armed forces and will report for duty as soon as called. Dr. Geis had moved to Graceville from Minneapolis but ten days before he was notified that he was to serve his country. He is the most recent addition to the staff of the Oliver Clinic there.

Dr. E. A. Thayer of Truman reported for military duty at Salt Lake City, Utah, October 13. He is a First Lieutenant in the Medical Corps of the United States Army.

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Dr. William Strunk of Northfield, director of health service at St. Olaf College, has received his commission as Captain in the Medical Corps and has reported for duty at Camp Custer, Michigan.

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Dr. D. D. Rempel of Lester Prairie has purchased the equipment of the late Dr. A. R. Johnson of Isanti and has located in the office formerly used by Dr. Johnson, for the practice of medicine.

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Dr. B. T. Bottolfson, physician and surgeon and former mayor of Moorhead, has been granted a fellowship at the Wills Eye Hospital, Philadelphia, where he is now engaged in study.

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Dr. Edward M. Baldigo of Red Wing has reported for military duty at Carlisle Barracks, Pennsylvania, following which he will serve with the Eighth Headquarters Command, Medical Department, at Dallas, Texas. He has the commission of First Lieutenant.

Dr. Gordon B. New of Rochester was elected president-elect at the annual meeting of the American Academy of Ophthalmology and Otolaryngology held in Chicago in October.

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Dr. A. V. Stoesser of Minneapolis was the guest speaker at the regular meeting of the Scott-Carver Medical Society held in Shakopee, September 22. His subject was "Allergy in Pediatrics."

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Dr. R. E. Johnson, assistant chief of staff of the Southwestern Minnesota Sanatorium at Worthington for the past six years, has resigned his post to take over the superintendency of Sunny Rest Sanatorium at Crookston.

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Dr. Claude F. Dixon of Rochester gave the Arthur Dean Bevan Lecture at a meeting of the Chicago Surgical Society in Chicago, October 1. His subject was "Anterior Resection for Carcinoma Low in the Sigmoid and Rectosigmoid."

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Dr. E. Graham Howard of Mapleton has been called into active service in the United States Navy and has reported to the Great Lakes Naval Training Station. He has the commission of Lieutenant in the Medical Corps.

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"Malaria" was the subject of the address given at the regular meeting of the Minnesota Pathological Society by Dr. L. T. Coggeshall of Ann Arbor, Michigan, at the Medical Science Amphitheater on the University Campus, Minneapolis, October 19.

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Dr. E. D. Risser of Winona has moved to Pasadena, California, where he will open offices and establish a practice. Dr. Risser came to Winona in 1921, being associated with the Winona Clinic five years before opening his own offices. He had been associated with Dr. G. L. Loomis since 1937.

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The Wheaton Hospital, Wheaton, Minnesota, was closed on October 1 mainly because of the shortage of nurses, it was announced. Drs. C. F. Ewing and A. L. Lindberg will continue their general practice as before, but major operative cases will be taken elsewhere. The hospital was first opened by Dr. Ewing in 1904.

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Installation of Dr. Thomas J. Kinsella as president of the Hennepin County Medical Society and the address of Dr. Willard D. White, retiring president, featured the annual meeting of the Hennepin County Medical Society, October 4. Dr. J. K. Anderson took office as vice president and Dr. A. E. Cardle as second vice president.

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Subjects presented at the Interdepartmental Seminar on the University Campus, Wednesday evening, October 27, included "A Practical Clinical Test for Organic Brain Damage" by Dr. Howard F. Hunt, of the Depart-

ments of Neuropsychiatry and Psychology, and "The Effects of Retrograde Degeneration on Neurone Activity" by Dr. Berry Campbell of the Department of Anatomy.

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Captain Alfred C. Meyer of the Army Medical Corps, former fellow in surgery in the Mayo Foundation, has been awarded the Silver Star for heroism and outstanding achievement in operating on wounded soldiers while under Japanese gunfire in New Guinea. It is reported that he is serving with the first portable hospital to be established by the Army.

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The Soudan Hospital, owned and operated for the past nine years by Dr. C. Gordon Watson, has been sold to the Lenont-Peterson Clinic of Virginia and will be operated by the Clinic with Dr. Leslie Laikola as resident physician. The Soudan Hospital has been in continuous operation for the past fifty-five years, being the oldest hospital on any of the iron ranges. Dr. Watson is leaving to serve in the Navy and following the war plans to establish a practice in Minneapolis.

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For outstanding work in the fight against tuberculosis, Dr. C. L. Sherman of Luverne received the Christmas Seal plaque from Dr. E. A. Meyerding, executive secretary of the Minnesota Public Health Association, at ceremonies held in Luverne, October 12. Dr. Sherman was instrumental in the founding of the Southwestern Minnesota Sanatorium and for the past twenty-eight years has served as president of the Sanatorium commission.

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Dr. W. J. Cochrane of Lake City was elected president of the Lake City Bank and Trust Company at a meeting of the Board of Directors held October 11. Dr. Cochrane has long been associated with the Lake City Bank and Trust Company, having been a member of the Board of Directors since 1907 and vice president since 1931. Dr. Cochrane retired from active medical practice a year ago. He succeeds Mr. H. F. Johns, recently deceased, as president of the bank.

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Dr. William W. Moir, former Stillwater physician, recently was awarded the distinguished service cross for his part in the Tunisian campaign. Although wounded in the head by a 20 mm. shell fragment and in the shoulder by machine gun fire from strafing airplanes, Dr. Moir, with the help of a sergeant and two company aid men, attended over twenty paratroopers injured when three American planes were shot down by enemy fighters. They stayed at their post twenty-four hours before being relieved.

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Dr. A. B. Stewart of Owatonna, oldest physician and surgeon of Steele County, quietly observed his eightieth birthday at the Colonial Hospital, Rochester, where he was a patient at the time. He has since returned to

Owatonna, where he has lived for the past forty-two years. Dr. Stewart was born in Hume, New York, September 23, 1863, and was graduated from the University of Minnesota in 1891. He came to Owatonna that year to practice his profession. He is now retired from practice.

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Dr. Walter P. Gardner, Superintendent of the Anoka State Hospital, Anoka, Minnesota, has resigned his post to return to private practice.

Upon his resignation, Dr. Gardner, former president of the Minnesota Hospital Association, and at present a clinical assistant professor of nervous and mental diseases at the University of Minnesota medical school, will become a partner of Dr. W. H. Hengstler of Saint Paul, who is also a specialist in nervous and mental diseases.

During his administration at the Anoka State Hospital, which has 14,000 patients, Dr. Gardner made a number of improvements for the care of patients in the institution.

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Dr. G. A. Hedberg, formerly Assistant Medical Director of Nopeming Sanatorium, was appointed Superintendent and Medical Director on July 1, 1943, succeeding Dr. A. T. Laird, who retired on that date to enter private practice in Duluth.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

AN INTRODUCTION TO MEDICAL MYCOLOGY. George M. Lewis, M.D. Member of American Dermatological Association, Inc.; Fellow American College of Physicians, American Medical Association and New York Academy of Medicine, etc.; and Mary E. Hopper, M.S., Research Fellow in Medicine, Cornell University Medical School. 342 pages. Illus. Price, $6.50, cloth. Chicago: The Year Book Publishers, 1943.

NERVOUSNESS, INDIGESTION AND PAIN. Walter C. Alvarez, M.D. Professor of Medicine, University of Minnesota (Mayo Foundation); Consultant in the Division of Medicine, The Mayo Clinic, Rochester, Minnesota. 488 pages. Price, $5.00, cloth. New York: Paul B. Hoeber, Inc., 1943.

THE COMPLEAT PEDIATRICIAN. Fourth Edition. Wilburt C. Davison, M.A., D.Sc., M.D. Professor of Pediatrics, Duke University School of Medicine, and Pediatrician, Duke Hospital, formerly Acting Dean of Department of Pediatrics, The Johns Hopkins University School of Medicine; Acting Pediatrician in Charge, The Johns Hopkins Hospital, Member American Board of Pediatrics, etc. 256 pages and Index. Price, $4.00, cloth. Durham, N. C.: Duke University Press, 1943.

THE MIND OF THE INJURED MAN. Joseph L. Fetterman, M.A., M.D. Assistant Clinical Professor of Nervous Diseases, Western Reserve University School of Medicine, Cleveland, Ohio. 260 pages. Illus. Price, $4.00, cloth. Chicago: Industrial Medicine Book Co., 1943.

TEXTBOOK OF PHYSIOLOGY. Eighth Edition. William D. Zoethout, Ph.D., Professor of Physiology in the Chicago College of Dental Surgery (Loyola University) and W. W. Tuttle, Ph.D., Professor of Physiology, College of Medicine, State University of Iowa. 728 pages. Illus. Price, $4.75, cloth. St. Louis: C. V. Mosby Co., 1943.

ORTHOPEDIC NURSING. Robert V. Funsten, M.D., Professor of Orthopedic Surgery, University of Virginia Medical School and University of Virginia Hospital School of Nursing, Charlottesville, Virginia; and Carmelita Calderwood, R.N., A.B., Consultant

(Continued on Page 1022)

in Orthopedic Nursing, National League of Nursing Education, New York; formerly Supervisor, Orthopedic Service, Iowa University Hospital, Iowa City, Iowa. Formerly Clinical Instructor in Orthopedic Nursing, Children's Hospital, Denver, Colorado. 602 pages. Illus. Price, $3.75, cloth. St. Louis: C. V. Mosby Co., 1943.

PERSONAL AND COMMUNITY HEALTH. Seventh Edition. C. E. Turner, A.M., Sc.D., Dr.P.H. Professor of Public Health in the Massachusetts Institute of Technology; formerly Associate Professor of Hygiene in the Tufts College Medical and Dental Schools; sometime member of the Administrative Board in the School of Public Health of Harvard University and the Massachusetts Institute of Technology. 585 pages. Illus. Price, $3.50, cloth. St. Louis: C. V. Mosby Co., 1943.

CLINICAL LABORATORY METHODS AND DIAGNOSIS. Third Edition. 2 Vols. R. B. H. Gradwohl, M.D., D.Sc. Dir. of Gradwohl Laboratories and Gradwohl School of Laboratory Technique; formerly Director of Laboratories, St. Louis County Hospital; pathologist to Christian Hospital; Director, Research Laboratory, St. Louis Metropolitan Police Department, St. Louis, Mo.; Commander, Medical Corps, United States Naval Reserve, ret. 2130 pages, Index 100 pages. Illus. Price, $20.00, cloth. St. Louis: C. V. Mosby Co., 1943.

DR. COLWELL'S DAILY LOG. Champaign, Illinois, Colwell Publishing Company, 1944. Price $6.00.

Many physicians throughout the country have found this volume in years past of inestimable value, not alone as a day book but as a complete record of office and personal income and expense. Spaces are supplied for deductible office and professional expense items, auto upkeep, taxes, interest paid, and collection fees, and pages have been added for recording quarterly Social Security deductions as well as other taxes, contributions, obstetrical cases, et cetera. As a matter of fact, this volume, in conjunction with a ledger, constitutes a complete financial record.

ATLAS OF OBSTETRIC TECHNIQUE. By Paul Titus, M.D., Obstetrician and Gynecologist, St. Margaret Memorial Hospital, Pittsburgh. 180 pages. Illus. Price $7.00. St. Louis: C. V. Mosby Company, 1943.

This book follows the modern trend in literature, that is, illustrations compose the greater part of the book, with a minimum of word description. Obviously, therefore, no discussion of indications for the various procedures appears. This would indicate that the book must be supplemented by a standard text.

A good part of the book is taken up by nonobstetrical material such as sterility studies and abdominal surgical procedures which might be carried on during pregnancy. These include such procedures as appendectomy, removal of ovarian cysts, et cetera.

Section IV on Version and Breech Delivery contains the best sequence of illustrations. One section illustrates various post-partum procedures such as sterilization operations, breast abscess and cautery of the cervix. On the whole, the illustrations are clear and concise. However, in a subject as varied and unstereotyped as obstetrics, only a partial picture can be obtained by this type of book.

S. C. MUELLER, M.D.

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